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Contents

Introduction

"Will My Heart Fail?" was chosen as the title for this book because it is a question that nearly everyone, at some time in his life, asks of himself or of his doctor. This is natural, since everyone knows that a person can get along fairly well without an arm, a leg, a lung, a kidney, a stomach, an eye or even a large part of the brain, but cannot live without the essential components of his heart pump. Moreover, heart disease is now the most frequent cause of death in the United States —a far more frequent cause than cancer—and this fact has been so well publicized that it would be extraordinary if most people did not at least occasionally fear heart failure. This book should reassure those who are concerned about their hearts.

At this point I should explain what I mean by heart failure. I do not mean cessation of heart action and death but, rather, the variety of disorders of the heart and blood vessels that cause these organs to fail in efficiency. This is a somewhat broader definition of heart failure than would be given by some doctors, to whom the term would mean such weakness of the pump

that that excess tissue fluid is retained by the body. Since heart failure in its earlier stages is not usually accompanied by such retention of fluid, nor is followed by sudden death, the use of the words heart failure should not be a reason for more concern than many other medical terms, such as influenza, a cold in the chest or gallstones. In other words, heart failure can be slight. If detected in its early stages it can usually be corrected by appropriate treatment.

Anyone who knows that he has suffered damage to his heart, whether in childhood, middle age or the later years, may fear heart failure. The teenager about to participate in competitive sports may have concern, even though he has considered himself to be in good health. A person whose heart is pounding because of worry or fatigue can scarcely avoid thinking of the possibility of heart failure. The executive faced with a new and challenging venture, and perhaps having seen even younger associates succumb suddenly to a heart attack, may feel fear. Many such fears should be dispersed if this book answers the question of its title.

This book begins with a background, that is, with a review of the structure of the heart and great vessels. Next it describes the way in which the circulation works, with a discussion of some of the complex controls involved. Then the weak spots in our hearts are described, for it is this sort of information, and much more, that allows

doctors to recognize the varieties of heart disease and treat them with understanding and success. There will be separate chapters on the chief *causes* of heart disease, in which the basic difficulties will be presented, as well as the types of treatment now available. What we owe to medical research will be emphasized, for advances in knowledge of heart disease have not occurred by accident, but as the result of painstaking experiments and observations by qualified investigators. Such research must continue if we are to find the answers to the problems as yet unsolved.

A damaged heart can improve if its owner has understanding, patience and purpose. It follows that a person's attitude toward his handicap is of greatest importance. Most of us fear the unknown. If our problems can be visualized clearly, we can direct our energies to meet the challenge, and thereby help our doctors to help us.

Chapter I

MECHANICS OF THE CIRCULATION OF THE BLOOD

THE HEART AS A PUMP

In this age of machines we are tempted to feel that such miracles as jet aircraft, electronic computers, rockets, and television are more complex and deserving of wonder than are the mechanisms at work in our own bodies. Such is not the case. The heart and blood vessels are a small but important part of the intricate systems which comprise the human body. The great complexity of the heart is demonstrated when attempts are made to construct machines able to perform its functions temporarily while delicate operations are performed within the heart kept at a standstill.

In their attempts to construct "heart-lung" machines, doctors can draw upon the large body of information about the heart and lungs that has resulted from years of research concerning their structure and functions. They are acquainted with many of the basic principles involved, and

have considerable information of a detailed nature concerning the operation of these principles. This present understanding is derived from research in chemistry, physics, and mathematics, as well as in the biological sciences. It has been their hope to be able to construct relatively simple heart-lung machines. In practice, however, no simple machine can reproduce the functions of the heart and lungs. Our bodies require that blood must be pumped by the heart to the tissues, within definite and critical limits of pressure, content of oxygen, carbon dioxide, and alkalinity, and with other subtle factors including the control of clotting, the type of blood appropriate for the individual, and the preservation and nourishment of living cells within the blood and throughout the tissues of the body. Some functions of the lungs include exchanges of oxygen and carbon dioxide, regulation of acid-base equilibrium, and the maintenance of body heat and fluid content.

It is not surprising, therefore, that artificial heart-lung machines have been successful to date in substituting for these organs for only brief periods of time. Although gratified with their initial successes, which have allowed lives to be saved, doctors are striving constantly for improvement. As yet they cannot construct a pump comparable with the heart, which is able to adapt itself to a wide variety of circumstances, to use fuel derived

from various foodstuffs, and to repair itself after major damage while continuing to function.

From the foregoing review of some of the functions performed by the heart it can be concluded that our hearts are indeed marvelous machines. Even the best man-made machines will wear out. This can also be said of all living things. It is estimated that an adult's heart beats about 100,000 times each day. A child's heart completes its complex cycle at an even faster rate. Each day the adult heart pumps about seven tons of blood through the blood vessels.

Many of you have had an opportunity to watch an automotive engine being built on an assembly line. To a bare framework are added various elements until the engine finally emerges, complete and ready to be tested. If it involves a new design, several such engines are allowed to run at top speed, while numerous tests are performed, and until they wear out. They are then torn down, in order that the weak spots can be examined and corrected. In a somewhat similar fashion doctors can find the weak spots in the heart. Some are readily identified after death when the heart is examined, as we keep in mind the functions performed by its various parts.

Structure or Anatomy

The human heart is a four-chambered pump, about the size of its owner's clenched fist, which

forces blood through elastic arteries simultaneously into two separate circuits. The weaker two *right chambers* of the heart receive blood from the veins of the body and pump it through the lungs. The *left* side of the heart receives oxygenated blood from the lungs and pumps it through-

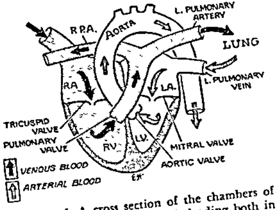


Diagram I. A cross section of the chambers of the heart and the great vessels leading both in and out. R. P. A.—right pulmonary artery. R. A.—right atrium. R. V.—right ventricle. L. A.—left atrium. L. V.—left ventricle. In the normal heart there is no admixture of venous and arterial blood. Prior to birth a shunt, the *ductus arteriosus*, allows blood to flow from the aorta into the left pulmonary artery. (Adapted, with the permission of the authors and the publisher, from C. S. Burwell and James Metcalfe, *Heart Disease in Pregnancy*. Little, Brown and Company, 1958).

out the arteries of the head, trunk, arms and legs (*see* Diagram 1) and back to the right atrium.

Each *side* of the heart can further be compared to two pumps shaped somewhat like rubber bulb syringes; an upper, thin-muscled *atrium*, discharging into a more powerful *ventricle*. The atria receive blood from the veins; the ventricles force blood into the arteries (*see* Diagram 2). As in the case of certain rubber bulb pumps, such as the one the doctor uses to take your blood pressure, valves are situated at the outlets to prevent backflow of blood into the atria and the ventricles after each contraction. These basic facts concerning the circulation of the blood were first described by an English physician, Sir William Harvey in 1628.

The heart might also be likened to a four-room house, designed for compactness and functional living. The two front rooms are the larger, and have thicker walls. These are the ventricles. The walls of the left ventricle are thicker than those of the right, since more muscular power is required to send blood throughout the body, as compared with the easier task of the right ventricle in supplying the lungs nearby. "Behind" the two front rooms are the smaller dressing rooms, the atria with thinner walls.

Surrounding each atrium and ventricle are firm walls of tissue which keep each room separate, so that blood must flow in its proper direc-

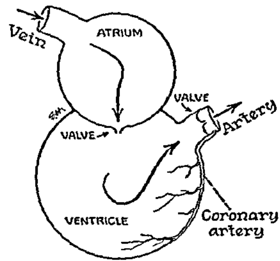


Diagram 2. The heart as a pump. Each half of the heart is similar to two rubber bulb syringes. A valve separates atrium from ventricle; another prevents backflow into the ventricle after blood is discharged by the ventricle into the artery. In this diagram the left side of the heart is shown, with a coronary artery arising just beyond the aortic valve. (This is a functional rather than an anatomical drawing. Therefore it does not correspond in detail to Diagram 1.)

tion, and admixture from right to left cannot occur. Flow from each back room (atrium) to its adjacent front room (ventricle) is through the roughly circular atrioventricular opening guarded by the valve to prevent backflow.

The outer wall of these rooms is composed of heart muscle. The contractions of this muscle pump the blood, and can be felt as the heartbeat. Arising from the top of the *left ventricle,* and coursing downward behind the heart in an arc is the largest and sturdiest blood vessel in the body. It is about the size of a small garden hose, and is called the *aorta* (Diagram 1).

The first branches arising from the aorta occur just above the *aortic valve* (*see* Diagram 2). These are the two *coronary* or "crowning" arteries which turn back to supply blood to the heart muscle of both the left and the right ventricle. They have numerous branches in order to supply the ceaseless demands of this, the most continuously active muscle in the body. It will be emphasized subsequently that these small coronary vessels cannot be filled with blood at a proper pressure if the aortic valve functions improperly due to injury or disease.

Close by the aorta, but emerging from the *right ventricle,* is the somewhat smaller *pulmonary artery* (Diagram 1). It courses through the arch formed by the aorta, and divides into two large branches which supply each lung. It is the only *artery* in the body that does not transport *oxygenated* blood. Coming from the right side of the heart, it carries "used" blood from the veins of the body to be oxygenated in the lungs

Blood enters the back rooms (atria) of the heart through six channels or veins. Two such

channels, draining *"used"* blood from all parts of the body except the lungs, enter the *right atrium. Oxygenated* blood from the lungs enters the left atrium through four smaller veins. These are the only *veins* in the body which transport oxygenated blood. (Ordinarily, veins carry blood with a low oxygen content.)

The Weak Spots

Certain mechanical disadvantages of this type of pump are apparent, despite its demonstrated efficiency. Since the pumping action results from contraction of heart muscle, it follows that this muscle is all-important. Fortunately it is the strongest and most efficient muscle in the body. The "hand" that squeezes these four bulbs is the *strength of the heart muscle itself. Indeed the* surgeon can supply this power temporarily with his hand if the heart should suddenly stop beating during an operation. Yet this muscle derives *its nourishing blood supply not from the blood* within the heart, but from two *"coronary"* arteries so small that each will hardly admit a pencil lead. As mentioned above, these arise from the large artery *(aorta)* which leads out of the left ventricle. If a portion of one or both of these coronary arteries should become obstructed or occluded, damage to heart muscle will immediately occur from lack of blood supply. *The dependency of heart muscle upon these small coronary arteries is a major weakness in our hearts.*

A second mechanical weak spot concerns the valves (Diagrams 1 and 2). These are four thin, veil-like structures which fall into place after blood is emptied by the contraction of the corresponding atrium or ventricle. Those guarding against leakage from the ventricles into the atria are anchored and controlled by fragile and delicate guy lines. The other two, guarding the outflow to the lungs and the aorta, are without such controls. They close because of the pressure exerted on them by columns of blood in the arteries as each ventricle completes its contraction. Because of their thin composition the valves inside the heart are subject to deformity and leakage as the result of a variety of disease processes. Thus diseases can eat away the substance of the valves or produce irregularity of their contours, which will render them *incompetent.* Also, weakening of the structures to which the valves are attached may result in such dilation or stretching that the valve will leak, producing a murmur that can be heard through the doctor's stethoscope.

It took many years of study and experimentation for doctors to learn the structural weaknesses of the heart. Now we are in the process of learning more about the chemical and mechanical secrets of its unusual efficiency, and are devising new ways for helping it to overcome the results of disease. Heart failure occurs when the heart muscle is so weakened that it is no longer

able to pump efficiently. From the foregoing *it is apparent that such failure is often associated* with improper function of the valves or with inadequate flow of blood through the coronary *arteries that supply the heart muscle.* Moreover, if blood supplied to the coronary arteries is inadequate in oxygen content, nutritional elements, *or number of red blood cells, then impairment* of the heart muscle can occur just as disastrous as if the coronary arteries were obstructed.

The Pump in Action

The foregoing description of the heart and blood vessels is necessary for an understanding of their basic structure. The heart is a pump that can rest for only a fraction of a second between beats: it cannot spare longer intervals for recuperation. Normally each heartbeat takes place in less than a second, there being about seventy beats per minute. During exercise or with excitement, the heart rate may normally increase to 150 or more beats per minute.

While the heart relaxes between beats, blood is flowing into both atria. Bright red blood, freshly *oxygenated* from the lungs, pours into the *left* atrium. Dark bluish "*used*" blood similarly flows into the right atrium. When the atria become full their muscles contract, forcing blood through the atrioventricular valves into the ventricles.

Less than a fifth of a second is needed by the atria to complete their contractions. Then the

ventricles contract vigorously, forcing shut the atrioventricular valves, which prevent backflow, and forcing open the valve of the aorta, and the pulmonary valve to the lungs. It is the forceful action of the left ventricle that we feel as the heart or pulse beat. With each beat the ventricle discharges about three ounces of blood into the aorta at a pressure of about 150 millimeters of mercury, sufficient to drive the blood through the furthermost tissues of the body. A like amount of blood is pumped to the lungs by the right ventricle, but under less pressure.

With the completion of ventricular pumping action, the aortic and pulmonary valves snap shut. The muscular contraction phase has occupied one-third of the cycle, the heart now rests for two-thirds of a cycle before these complex events are again repeated. Although these cycles generally occur at an average rate of about seventy per minute while at rest, the rate of the resting heart in well-trained athletes is usually slower, it may be only forty. Some persons, however, may have a resting heart rate of more than 100 without suffering any disadvantage. During exercise the heart automatically increases its rate to 150 beats or more per minute; this is the expected response to the need of the muscles for more blood.

To maintain this cycle of repeated heartbeats at a speed appropriate for the needs of the body there is a series of sensitive nerve signals, called

reflexes, arising in various parts of the heart and circulation. These act to control the heart rate and the amounts of blood received by and pumped out through the chambers of the heart.

Red and Blue Blood

As we have already learned, within the normal heart there is no admixture of the used (blue) blood from the veins with the red (oxygenated) blood from the lungs. Used blood courses through the right atrium and ventricle. In the lungs oxygen is added to give the blood its bright red color (*oxyhemoglobin*). Thus the left atrium and ventricle normally pump only this bright red blood, which has been oxygenated in the lungs.

Babies are sometimes born with abnormal openings *inside the heart, as between the atria,* or between the ventricles. So far we do not know exactly why faults of development occur. There *is evidence that genetic factors, infections, and* other influences can operate during pregnancy to produce such abnormalities. In such infants, *due to admixtures of "red" and "blue"* blood there may be blueness of the lips, fingernails and toenails. Since heart muscle can function properly *only in the presence of sufficient* red *or* oxygenated blood, heart failure is more likely to occur when there are defects that produce blueness. *Moreover, other tissues of the body* besides the heart may not develop properly or

function normally when there is oxygen lack. The case of Jimmy is an illustration.

Jimmy was born a "blue baby." At the age of twelve he was referred for consideration of operation. In addition to having blueness of his lips and nails, he was thin and weak. After even mild exercise he would have to rest. By means of tests, to be described later, it was learned that the chief cause of his blueness was an abnormal opening between his two ventricles. He and his parents consented to a corrective operation. When he returned for an examination three months after operation he was no longer blue, he had grown *two inches* in height, and he could play baseball with his classmates for the first time because good red blood had been made available to his needy tissues. Jimmy is a striking example of the necessity for the left and the right sides of the heart to remain separate.

Control of the Pump

As we have seen, the human heart keeps blood flowing simultaneously through two entirely different circuits, one of which includes the lungs, the other the remainder of the body. The hearts of some of the lower animals do not permit this clear separation of "used" and oxygenated blood. Like the engine of an automobile with multiple cylinders, your heart has delicate and precise controls to maintain the proper speed, timing, and

pressures needed to ensure an adequate flow of blood to a variety of organs such as the brain, lungs, kidneys, and muscles—including the heart muscle itself. It must be responsive to the unusual needs of any part of the body, while at the same time maintaining sufficient blood flow throughout. Usually the heart responds efficiently to these various needs.

Automatic communications systems (reflexes) act like complex electronic devices to send messages to and from the heart. Occasionally the circulation may fail to function with complete success when the body is under unusual stress. The brain, for instance, may be cheated in competition with other organs following great athletic effort, or when blood pools in the lower portions of the body during prolonged standing, as for a military inspection. Under such circumstances a brief attack of fainting may occur. Although such an episode may seem alarming, it is nature's response to the law of gravity as it affects the individual who is standing erect. When he falls to the ground in a faint his body becomes horizontal instead of vertical, blood flow to his brain is quickly restored, and he returns to consciousness.

One of the most exciting phases of medical research has been the discovery of an increasing number of the automatic controls that govern the activity of the heart in sickness and in health. Although these computor systems consist of

electrical impulses, such reflexes may be initiated by changes in pressure, temperature, or the chemical composition of the blood. A few examples will serve to illustrate the functions performed by these controls:

Our bodies may respond to infection with fever. This occurs whether the infection is widespread, as with influenza, or localized as in acute appendicitis. Blood vessels about the area of infection (appendicitis), or throughout the body (influenza) tend to relax, enlarge, or *dilate*. In the case of a general infection there is often an initial period of contraction of small blood vessels and a sense of chilliness, or even a shaking chill. Subsequently messages are transmitted to the control center (thermostat) in the brain which governs the body's temperature, and this becomes reset to allow fever to occur. Responsive to fever there occurs further dilatation of blood vessels. Other reflexes signal the timer (*pacemaker*) in the heart to put out more blood in order to fill the dilated blood vessels. Accordingly the speed of the heartbeat is increased. In addition, through further complex controls within the heart, a larger volume of blood is pumped with each contraction. Additional blood thus becomes available to combat infection and to aid in the removal of poisonous waste products. In the case of appendicitis, more white blood cells will enter the area of infection to destroy bacteria. This sequence of events has been discovered after

painstaking studies in both animals and humans. It involves a number of reflex relay stations situated in the brain, spinal cord, heart, and blood vessels. All of this lies behind the familiar observation that when a person has a fever his pulse rate will increase.

Similar related events explain why we are likely to feel drowsy after a large meal. Animals are wise enough to yield to this feeling by taking a nap. Humans may fail to heed the wisdom of the body, in the presence of heart disease, and may exert themselves during the process of digestion. Following an average meal the output of the heart can be shown to increase by as much as forty per cent. The greatest increase is observed within an hour after a meal, but a return to the fasting level of output does not occur until about two hours later. It can also be demonstrated that, during digestion, there is a sharp increase in the amount of blood sent to the abdominal organs. This facilitates the digestion and absorption of food. In addition, there is a considerable increase in the flow of blood to the skin during the process of digestion. Hence we may expect a man to have a flushed face after a hearty meal. Thus, if the output of the heart is increased by forty per cent during the digestion of a meal, and if most of this augmented blood flow goes to the abdominal organs and to the skin, is it any wonder that the brain may signify its relative lack of blood by becoming sluggish at

this time? Or, from another point of view, does it not seem reasonable for the person with heart disease to eat lightly and to avoid physical activity for an hour after eating? To exercise during digestion is to increase the need for the heart to work in excess of the forty per cent increase occasioned by the digestive process.

How has it been possible to measure the controls that govern the heart's action? Concerning this question many reports and books have been written. It is in order here to give some indication of how such information was obtained. First, it was necessary to understand the structure of the heart, blood vessels, and other organs in the body which function together. This required centuries of the study of both man and animals. It became apparent that simple creatures, like the earthworm and the dogfish, have relatively simple hearts and circulations. Conversely, higher animals have hearts with more specialized developments, having two, three or four chambers.

The careful dissection and study of the anatomy of dead bodies of men and animals has been pursued for many centuries, the earliest records dating back to an Egyptian, Imhotep, who lived about 3,000 B.C. With the discovery of the microscope, previously invisible structures could be identified. In order that disease could be understood and corrected, the next step was to dissect the living bodies of animals. Finally

Sir William Harvey solved the riddle in 1628, showing the course taken by the currents of blood in passing through the heart. Tremendous as was this discovery, however, it revealed only the basic facts of structure and function. It was as though a visitor from Mars to this planet had come upon an automobile in a junk yard, and was trying to understand its function after the carburetor, wiring and accessories all had been removed. The careful observation of the exposed heart of a living animal was not sufficient to yield many secrets. It became necessary to set up experiments of a purposeful nature, and to borrow the tools of chemistry and physics in order to understand how the heart works.

In 1727 an English clergyman, Stephen Hales, first measured blood pressure by thrusting a tube into the artery of a horse. This may well have been the beginning of precise measurements of the circulation. Since that time many types of apparatus have been devised for measuring pressures in blood vessels both large and small. With the aid of a microscope, delicate tubes can be introduced into the capillaries of the skin, kidney, or other organs. Using a fluoroscope or x-ray machine for guidance, it has been possible to direct small flexible tubes or catheters into the chambers of the heart, and into most of the blood vessels of the body. The discovery of chemicals that outline blood vessels in sharp contrast on an x-ray film has allowed doctors to study the

circulation in various parts of the body, notably the heart, brain, and kidneys.

It is implicit in the scientific method that experiments be designed in advance to test the response or responses of the body to a well-defined stimulus—chemical, electrical, or mechanical. Our task has been to gather as much information as possible from each experiment, and to record the circumstances of each trial so accurately that others may perform the same test to prove or disprove the initial findings.

A single experiment to test the effect of stimulating a particular nerve that might affect the heart may require the following apparatus: a machine for delivering accurately graded electrical shocks; devices for recording continuously the blood pressure, respiration, heart action (electrocardiograph); catheters to be inserted in the heart, arteries and veins for measuring not only pressures, but also to allow removal of blood samples for chemical analysis and calculation of the output of the heart. Special equipment for measuring the concentration of radioactive chemicals may also be needed in such elaborate experiments on animals.

Through such experiments it has been possible to isolate the regions from which the heart's actions are controlled through electrical (reflex) or chemical governors. It should be apparent that some of them are situated remotely, in distant parts of the body, in order that the activity of the

eart can change with the needs of the body as whole. Important centers for the control of blood pressure have been found to lie in the arch of the aorta and in the carotid arteries, i.e., those in the neck. When blood pressure rises or falls, these centers send electrical messages to arouse activity in the heart and blood vessels which will tend to return the blood pressure to its original level. Within the right atrium lie similar nerve-endings which govern the heart rate and the volume of blood ejected with each beat. This mechanism allows the heart to become neither too full nor too empty, with variations in the amount of blood entering the right heart. The control of the pump is thus a matter of delicate checks and balances. Although we have learned a great deal concerning the mechanisms involved, much remains to be learned.

These automatic controls are able to compensate in large part for disease and deformity of heart valves, or for abnormal openings between atria and ventricles. Only when heart muscle has been damaged, or subjected to prolonged stress because of valvular disease, do these adaptations fail Then heart failure may occur.

Some Consequences of Heart Failure

Heart failure affects many parts of the body. When the pump fails, blood tends to stagnate in various organs, such as the lungs, liver, and kidneys. The kidneys may lose their ability to

rid the body of salt, water and various waste products. With congested lungs, oxygenation of blood is impaired so that blueness of the fingernails and shortness of breath will appear. When the liver becomes overfull of blood the victim may be conscious of a painful swelling in the upper abdomen. Puffiness of the feet and ankles may be an early sign of the inability of the body to rid itself of excess tissue fluid, but can also be due to causes other than heart failure.

It is not uncommon for one side of the heart to show more marked signs of failure than the other. Doctors may speak of "right-sided" or "left-sided" failure. If it is assumed that the side which fails allows pressure to be built up behind it, the consequences of its failure can be understood and demonstrated. In brief, if the right side fails there will be an increase in pressure within the veins of the body, all of which empty into the right atrium. The veins of the neck and arms will stand out like cords. The liver, unable to drain blood through its veins, will become enlarged and tender. In keeping with increased pressure in the veins of the legs, fluid may accumulate under the skin. The swollen legs and ankles due to heart failure are called *edematous,* or filled with edema fluid.

A glance at Diagram 1 will remind you that if, because of failure, blood does not enter and leave the left heart freely, the lungs will bear the brunt of difficulty and will become overfull of

stagnant blood. This state of affairs can occur when there is such strain on the left heart that it can no longer pump efficiently. Common causes of left-sided failure include high blood pressure and deformity of either the mitral or the aortic valves. The symptoms of left-sided failure arise chiefly from the lungs, with varying degrees of shortness of breath, cough, and even blood-tinged expectoration.

Having pointed out that failure may involve one or the other side of the heart, I must now hasten to add that only seldom is heart failure *confined* to the left or the right side. It may begin primarily as a result of strain and damage involving one side. In its more advanced stages, however, all four chambers are likely to be involved. It is a medical axiom in this regard that, "The most common cause of right-sided failure is left-sided failure."

These consequences of heart failure indicate how dependent the rest of the body is upon the heart. Since blood flow through the coronary arteries is adequate only when heart muscle contractions are properly forceful, a vicious cycle can occur during severe failure, when the weakened heart literally fails to nourish its own muscles.

New Hope for the Heart

Following many studies an understanding of heart failure, as outlined above, has developed.

Through related discoveries it is now possible to measure pressure and blood-gas content in even small arteries, and to calculate the degree of circulatory failure present in various parts of the body. Small flexible catheters can be threaded into blood vessels, and within all chambers of the heart, with but little discomfort to the patient. Using precise measurements, the effectiveness of various drugs can be evaluated. Through studies of this sort, the treatment of heart failure is constantly being improved and simplified.

The case of Mr. M. B. will illustrate how heart failure can be treated successfully. At the age of eighty he had become aware of increasing shortness of breath, a tight wheezing cough that was most severe at night, and swelling of his ankles so great that he could not wear his shoes. Accordingly he found it necessary to sit in a chair for most of the night. He shuffled about in loose slippers. Feeling that he was paying the price of becoming elderly, he despaired of his life and became despondent.

Upon examination it was felt that he was suffering from heart failure due to hardening of the arteries, or *arteriosclerosis.* His blood pressure was normal for one of his age. There was no evidence of important complication such as diabetes or kidney failure. Accordingly, he was encouraged to follow a few simple instructions, and to take a new form of a drug (aminophyllin)

related to caffeine (coffee). A month later he reported from his vacation camp that he felt quite well. His shortness of breath, cough, and the swelling of his ankles had disappeared. He was fishing and enjoying trap shooting in a fashion he had not thought possible to regain. His depression had lifted.

Although in this instance the treatment could be simplified and could be explained during a single office visit, these practical suggestions with respect to medication were based on a long background of experience. This drug was chosen for Mr. M. B. because it has the dual action of increasing the output of the heart and of clearing congested lungs. These actions had been confirmed abundantly in trials upon both humans and animals. Unfortunately, it cannot be given by mouth in an effective dose without upsetting the digestion. It is well tolerated, in dilute solution, when given by vein, but this route was not practical in the case of Mr. M. B. A new solution of the drug, for use as a rectal instillation, was the form in which it was suggested for this elderly gentleman. Tests of this solution, given to patients with either asthma or heart failure had shown it to be both safe and effective. Its usefulness in the case of Mr. M. B. can hardly be disputed.

Elastic Tubing-Arteries and Veins

We have already seen that it is impossible to understand the heart as a pump without considering the passages leading into and away from it, together with the intricate ramifications of the nervous system which allow the circulation to respond to the changing needs of the body during rest or exercise, in health or in illness.

The strong, elastic tubing leading from each ventricle comprises the arteries. The aorta, leading from the left ventricle, is larger and stronger than the pulmonary artery, since it must carry blood under greater pressure to all parts of the body. As mentioned earlier the pulmonary artery carries blood under lesser pressure to the lungs close by the heart.

The final network of vessels, conducting blood between arteries and veins, is composed of microscopically small and thin-walled channels known as capillaries. It is in this network that the red blood cells surrender oxygen to the tissues. Various other nutriments are delivered through the capillaries. Here, also, the red cells pick up carbon dioxide to be borne back to the lungs and discharged. Other waste products, likewise, are picked up in the capillaries, to be filtered out by the lungs, liver, or kidneys. Upon looking through a microscope one can appreciate the ceaseless activity of the capillaries and [illegible]

blood vessels. With such a preparation it is possible to follow the movement of individual cells in the blood or walls of the capillaries. One can also observe changes in the capillary tree resulting from the stimulation of nerves, or the introduction of chemicals either locally or into the blood stream. The blanching of the skin that follows a sudden fright can be seen to be associated with a cessation in the flow of blood through the capillaries. A number of scientists have spent the whole of their professional lives observing and recording the structure and functions of these delicate vessels.

Leaving the capillary network, blood returns to the right atrium through the relatively thin-walled veins. In some areas, such as the head of the human (or giraffe!) this venous return is largely enforced by gravity. Relative pressures within the circuit also play a part, aided by the pumping action of the lungs, muscles, and heart. That veins are both thin and elastic can be demonstrated by watching the veins on the back of your hand, full and bulging as the hand is held hanging down, or small and barely visible as the hand is lifted above the shoulder, and thus above the level of the right auricle.

By contrast, an artery such as the one which courses just above the elbow on the inside of the arm can be felt to pulsate and to have thick walls. It is common knowledge that when an artery is cut or torn, bright red blood will spurt with

each beat of the heart. To stop such bleeding a tourniquet must be applied between the heart and the injured area, and with pressure in excess of the patient's blood pressure.

Both veins and arteries are controlled by parts of the same reflex nerve system that governs the heart's rate and force. If more blood is required in one part of the body, as in the stomach during digestion, the arteries, veins, and capillaries connecting them will relax in that area to allow more blood to flow in. To compensate, blood vessels in other parts of the body may contract to aid in forcing blood to the region of need. This type of change occurs automatically and instantaneously when we get out of bed each morning. Through reflex control, contraction of arteries and veins occurs in the legs, making more blood available to the brain, heart and other organs when we stand erect. When these reflex changes do not take place, due to impairments in the nervous system, the individual may faint whenever he stands quietly. This occurs because the blood vessels in the legs do not respond as efficiently as usual. Indeed, they form a pool where blood is trapped away from the rest of the circulation. For this type of failure of the circulation the heart is not at fault.

A patient who was unable to stand quietly without fainting was first examined in our hospital in 1940. Not only was it possible to demonstrate that due to faulty nerve control, the

blood vessels in his legs did not contract when he stood, but also that the related nerve supply to his heart was interrupted. As a result his heart rate remained at a slow, fixed level. With exercise or during quiet standing, no increase in heart rate could occur. It would have been to his advantage if his heart rate could have increased, to compensate for the sharp fall in blood pressure that occurred when he stood quietly. He had learned to keep his legs moving at all times. If engaged in conversation while standing he would shift his weight from one leg to the other, thus utilizing the pumping action of his leg muscles to force blood in the veins of his legs back toward the right heart.

During cold weather this patient was able to compensate fairly well for the paralysis of the sympathetic nerves which normally act on the heart and blood vessels. In hot weather, however, his small blood vessels would dilate as a result of the heat and he would faint and have convulsions if he ventured into a warm room. Drugs seemed to be of little help in counteracting the tendency for his blood pressure to fall. He has attempted to avoid his attacks by remaining in the basement or in an air-conditioned room during warm summer weather. He is a striking example of the important part played by the sympathetic nervous system in the control of the heart and circulation.

To summarize, it is necessary that arteries and

veins be elastic and under control of nerve reflexes, in order to perform their functions of maintaining blood pressure and of transporting blood. They should also remain free from obstruction. Within the blood-forming organs are produced complex chemicals which allow clots to form when needed to control hemorrhage, but tend to prevent their occurrence when they might obstruct healthy arteries and veins. More will be said of abnormalities of arteries and veins as they pertain to the problem of heart failure. Their proper function is as important as that of the heart itself.

Chapter II

WHY DO I FEEL THIS WAY? THE DIAGNOSIS OF HEART DISEASE

As medical students advance in the study of various diseases they are prone to recognize that they themselves have felt some of the discomforts or *symptoms* described, and may wonder if they, too, may not have such diseases as cancer or tuberculosis. Having mentioned to you some of the symptoms of heart failure, it is now my obligation to help you in your quest to discover whether or not your feelings might be due to actual heart disease.

It has been pointed out that shortness of breath, blueness of the fingernails, and swelling of the ankles can be evidences of heart failure. Rapid heart action may be in the same category. More commonly, however, such palpitation is an expression of nervousness. Or pain about the heart can occur if the heart muscle is receiving insufficient oxygenated blood, as a result of obstruction to flow through the coronary vessels or other causes. Does the occurrence of one of these types of discomfort necessarily indicate

serious heart disease? The answer is "no." Any one of them can be due to disease outside the heart, and some of them to nothing more than nervous tension and fatigue.

If such symptoms are troublesome, you should see your doctor for an examination, since he is trained to help you by interpreting your symptoms or feelings in relation to what he finds as he examines you. Better still, you should have an annual examination even though you feel quite well, since heart disease can exist with but little discomfort.

The diagnosis of heart disease is like solving a crossword puzzle or a detective story. A number of answers are possible, but you and your doctor are interested only in the correct one. To arrive at the proper diagnosis he will ask you the exact circumstances under which you are likely to notice various symptoms, and details concerning their characteristics. These have especial significance to him. He is dependent upon your observations and descriptions of your feelings to guide his examination and selection of special tests to be performed. Among other things, he must determine whether your distress is due to a damaged heart, to disturbance in its function, or to a disorder located elsewhere in the body. He will want to know your past record of health and sickness, and your family's, together with your habits of living and the worries to which you are subject.

The importance of having a full story of your symptoms cannot be overemphasized. Let us start with the time when you first realized all was not well. Under exactly what circumstances did you make the discovery? Did morning headaches drive you to your doctor, who discovered that your blood pressure was too high? Did you have scarlet fever or rheumatic fever as a child? Was that nasty feeling of tightness under your breastbone and down the inside of your arms first noticed as you hurried to the bus stop on a cold morning after a hearty breakfast? What happened next, and after that? How do your symptoms at present compare in severity and frequency with those first warnings?

Now you're getting the point! What you tell us is just as important for the diagnosis as what we find when we examine you. But it must all hang together. If you tell us you're short of breath and can't walk up a flight of stairs, but if you play golf with ease and sleep on only one pillow, then we suspect you may be more anxious than short of breath, and we may try hard to find out what business or domestic problems you ar facing. Contrarily, if you had rheumatic feve as a child, have an *enlarged* heart and som *signs of failure*, but feel well while doing sede tary work you may be lucky, but a caref. program of treatment should nevertheless be started.

If he suspects heart disease your physician

will pay particular attention in his examination to your weight, pulse, blood pressure, the condition of your blood vessels, the size of your heart, the nature of heart murmurs, and evidences of fluid retention, if present. At the same time he will be searching for disorders other than those involving the heart. He will probably perform some tests on your blood and urine. If in doubt about the best course of action, he may ask you to visit a heart specialist or internist of his choice for more intensive studies, such as an electrocardiogram and chest x-ray, or a fluoroscopic study of your heart and lungs.

At this point it seems desirable to describe some of the equipment and tests doctors use in the evaluation of patients with known or suspected heart disease. As a result of continuing research, new tests are coming into use each year, but this description will be limited to those in most common use at present.

Laboratory Tests

In performing a blood count, blood is drawn either from a vein in the arm or from a finger tip. By using small measuring tubes, a precise quantity can be withdrawn and mixed with a known amount of diluting fluid. After thorough mixing the fluid is then placed in a glass chamber of standard dimensions in order that the number of both red and white blood cells can be counted. A smear of blood can also be placed on a

ide and stained, in order that the proportion f various types of white blood cells can be counted. The red pigment (*hemoglobin*) in the blood is estimated by comparison with color standards. The proportion of red blood cells, in relation to the rest of the blood, is determined by whirling blood in a centrifuge so that the heavier red cells settle in the bottom of a small, measured tube called a *hematocrit.*

The blood count can be of considerable assistance in the diagnosis of heart disease. Too few white blood cells may indicate an inability to combat infection. Too many white cells may mean that the body is responding to an infection, which might not otherwise be evident. Those parts of the blood count dealing with the hemoglobin and red cells are of importance in determining whether these elements are unusually sparse (*anemia*) or abundant (*polycythemia*). An abnormality in either direction may result in impaired oxygenation of heart muscle, giving rise to shortness of breath or pain about the heart. These symptoms may disappear when the hemoglobin and red cells are brought to normal by appropriate treatment.

Examination of the urine includes measurement of its acidity, specific gravity or density, content of protein and sugar, and its content o cells as observed under a microscope. Thes examinations not only help the doctor to diagnos disease of the kidneys, but also may give ir

portant evidence of disease elsewhere in the body, such as diabetes. To exclude diabetes positively, however, it may be necessary to perform chemical determinations of sugar in blood drawn from a finger tip or arm vein. Likewise, if examination of the urine suggests kidney disease, further measurements of the function of the kidneys may be necessary. These will involve special chemical examinations of both blood and urine.

In taking your blood pressure the doctor uses two instruments, a *stethoscope* and a *sphygmomanometer.* The stethoscope is a simple device the ear tubes of which fit into the doctor's ears and allow him to concentrate upon the sounds produced beneath the other end of the device. As he takes blood pressures, he listens over the artery that courses on the inside of the elbow joint.

The sphygmomanometer contains three basic elements: (1) a dial or column of mercury from which actual pressures can be read, (2) an inflatable cuff which wraps around the arm and can be filled with air, and (3) a rubber bulb by which air can be pumped into the cuff, fitted with a valve to prevent leakage (for comparison *see* Diagram 2).

It is desirable that the patient be as relaxed as possible during the taking of blood pressures. He should not crane his neck to see the dial, especially since he cannot read his own pressure accurately by this maneuver. Furthermore, he

cannot be thoroughly relaxed while making such an attempt. The sounds heard through the doctor's stethoscope are the true measures of blood pressure levels. After pumping air into the cuff to a high pressure, the doctor listens as air is released. As blood begins to flow through the artery, sharp sounds are heard with each beat of the heart. This marks the upper (*systolic*) blood pressure. When the sounds fade out completely, the lower (*diastolic*) pressure is reached. Hence the doctor, in recording blood pressures, will record two numbers, such as 120/80.

Another instrument used in the examination of patients with heart disease was developed to allow one to observe the inside of the eyeball. It is called an *ophthalmoscope*. With this device doctors can see small arteries and veins, lying almost naked and devoid of covering material. In this location hardening or narrowing of small arteries can be seen, together with changes which may lead to the discovery of such important disorders as diabetes or kidney disease (*nephritis*).

The *electrocardiogram* is a useful device for recording the electrical activity of the heart associated with each contraction. Wires are fixed into electrodes, attached to both arms and legs, leading into a control device for recording the currents. Another electrode picks up currents from over the body of the heart. Although patients are prone to compare this machine with the electric chair, it should be emphasized that

the machine serves only to amplify the action currents produced by the heart and does not produce any sensation or discomfort. If an abnormal electrocardiogram is found, it may be of considerable assistance in pointing out an area of damage or strain in the heart muscle. It should also be known that a normal electrocardiogram does not positively exclude damage to the heart. Often a number of electrocardiograms must be taken to evaluate adequately the condition of the heart muscle. This machine is of great value in clarifying the nature of various irregularities of rhythm. It is of relatively little importance in helping us to understand the exact nature of valvular disease of the heart.

Finally, the shape and size of the heart can best be determined with x-ray equipment. This can be done with films for a permanent record, or the doctor may place the patient behind a *fluoroscopic* screen in a dark room and trace an exact *orthodiagram* of the heart and great vessels, since they appear in sharp contrast with the adjacent air-filled lungs.

Informed by such examinations, your doctor is able to tell you with assurance whether or not your symptoms are due to heart disease and what your course of action should be. Thus, in place of worry and uncertainty you can have reassurance and a definite program tailored to your personal needs. If you must accept a verdict of some damage to your heart, you will want

repetitions of these examinations at intervals frequent enough to keep you informed of your progress.

What Kind of Heart Disease Do I Have?

Let us assume that your doctor has completed his examination and has found definite evidence of abnormality of your heart and circulation. It may help you to cooperate with him if you first understand how he fits together the pieces of the puzzle into a pattern that allows him to counsel you in a wise and practical fashion.

Many years ago the New York Heart Association proposed a five-point approach to the diagnosis of heart disease. Subsequently this was adopted by the American Heart Association and it is now in use throughout the United States. According to this approach, the specialist in heart disease will answer these five questions as he makes his diagnosis:

The Doctor's Evaluation
A Five-Point Quiz

1. What is the cause, or causes?
2. Is the heart enlarged or damaged?
3. Type of irregularity of heart action?
4. How marked is the patient's feeling of disability?
5. How much restriction of activity should I advise?

This five-point approach is your guarantee that you will have the benefit of careful study before recommendations are made concerning treatment.

The commonest causes (point one) of heart disease that your doctor will consider include overweight (*obesity*), hardening of the arteries (*arteriosclerosis*), high blood pressure (*hypertension*), rheumatic fever, developmental effects prior to birth (*congenital*), and disturbances of metabolism, such as are associated with goiter and diabetes. There are many other rarer causes which need not be considered here. Even if no actual heart disease is found during the doctors' examinations, this five-point quiz is completed for all patients examined in a heart clinic or in the office of a heart specialist. When such a systematic approach is employed, in connection with his medical knowledge, it usually is not difficult for the physician to determine the likely cause of heart disease in the individual case.

The question of heart size (point two) is a knotty one, of importance to both doctor and patient. The knowledge that his heart is enlarged may lead its owner to fear that he may be on the verge of heart failure. This is rarely the case. Sometimes, where there are certain defects in the valves, the heart appears to function more efficiently if its chambers are enlarged or thickened. In case of doubt, careful examinations by x-ray may be required to determine whether or not

the heart is actually enlarged. It may not be enlarged, but only tilted, or shifted to the left. If an increase in size is clearly demonstrated, repeated periodic examinations will allow the doctor to learn whether or not progressive changes, for better or worse, are occurring.

For several years a patient (who is also a physician) has requested annual examinations because there appeared to be some enlargement of his heart when he applied for insurance several years ago. Because of this he was not allowed to purchase protection at standard rates. Through utilizing fluoroscopy it is evident that his heart is not enlarged, although at first glance this might seem to be the case. His chest has an unusual shape, being "flat" or unusually narrow from front to back. In association with this unusual shape his heart is situated to the left and is tilted upward at its apex. As seen through the fluoroscope from various views the shape and the volume of his heart appear normal. This physician wisely requests periodic examinations "for the record." No change in heart size has been detected during the past four years. His son also has this unusual configuration of his chest and heart. Neither, of course, can be said to have heart disease.

In answering the second question in his quiz about you, your doctor will also record any evidence of structural abnormality other than

an enlarged heart. Quite commonly, a heart murmur is heard, particularly when the heart rate is increased with exercise. As heard through the doctor's stethoscope this sound may have, as its name suggests, a murmuring, blowing, scraping or rumbling quality. Such sounds are usually produced by jets or eddies of blood, as when the blood stream passes through a narrow orifice into a chamber or larger vessel. Often such murmurs do not signify valvular leakage or damage. In such instances, although the presence of the murmur is recorded, no diagnosis of an abnormality is made. Let us reach a clear understanding about this matter: *If you or one of your loved ones is found to have a heart murmur, its presence does not necessarily mean heart disease.*

What Is the Diagnosis?

At this point you have probably already guessed why the five-point quiz is of substantial aid to doctor and patient alike: It helps us to answer the practical questions, "What does all this add up to?" and "What's to be done about it?" Thus, in a patient with no symptoms, and nothing to be found save a trifling murmur, we may conclude that no heart disease is present, and the answer to the final three questions of the quiz is easy. Or, if a patient has no background of rheumatic fever in childhood, and no symptoms of disease

of his heart or other organs, the finding of a slightly abnormal electrocardiogram alone will not justify a diagnosis of heart disease.

On the other hand, let us suppose that a child has previously suffered an attack of rheumatic fever, and has a murmur indicating valvular damage. If he is without symptoms, able to perform all his usual activities without discomfort, shows no evidence of active rheumatic infection, no signs of heart failure, and no enlargement of his heart, he and his family can usually be reassured. Again the last three answers to the quiz will be favorable, but re-examinations probably will be suggested.

If the picture is darker, however, as it is when there is considerable enlargement of the heart, shortness of breath, or other symptoms and signs of failure, and irregular heart action, then the doctor must recommend restriction of activity, medication, or even operation in certain instances. With respect to operation, it will be advised in light of the patient's age, the severity of his heart disease, the course likely without operation, and knowledge of the results of the operation in many other persons with similar valvular disorder. Here again, we are giving the patient the benefit of our total knowledge not only about him but many other similar cases as well.

Irregularity of the heart (point three) or rapid heart action is likely to raise the familiar question, "Will my heart fail?" Here again the five-

point questionnaire will give the answer. If you have no other evidence of heart disease, your heart may bump, thump and jump in a very irregular fashion yet will do itself no harm under these circumstances even though it may be hard for you to believe this. Often it is desirable to have an electrocardiogram taken during the irregularity to determine its precise nature. If its antics disturb you too much, your doctor can usually prescribe medication to quiet your heart. He will probably urge you to smoke less, drink less tea, coffee and cola, and get more rest. These are not just routine suggestions; they are of practical importance. If you doubt this, make yourself follow the rules for several weeks. You'll be pleased by the results. Furthermore, don't take the poison and try at the same time to get relief from an antidote, as did one patient who took large doses of sedatives throughout the day to quiet his nerves and his heart but was also drinking eight cups of strong coffee daily!

If your heart is basically sound, we have seen that irregularities are disturbing but not dangerous. A few rare irregularities are more apt to occur when the heart is damaged. When they do, the physician can usually bring them under control. On the other hand, even the damaged heart often can tolerate rapid or irregular action: Following severe damage to his heart as a result of a coronary occlusion, a man of sixty developed a persistently rapid heart rate. For ten

days after his heart attack this man's heart continued to beat at 180, despite the use of several drugs usually effective in this situation. Each day it was feared that his heart would go into acute failure. It didn't. The rate dropped back to eighty on the tenth day. He made a slow but otherwise uneventful recovery.

Having carefully estimated the degree of disability recognized by the patient (point four), the doctor's next step is to weigh the patient's discomfort against the evidences of heart disease that have been found upon examination. This will include an interpretation of the various laboratory tests described. Having considered all of this information, the doctor can determine whether or not his patient is living within the limitations of his heart's impairment and what advice regarding activity (point five) he shoul offer. Sometimes a person with heart disease i pulling his punches and is doing less than h should. The doctor's final diagnosis, in the ca of a patient with rheumatic heart disease, mig be as follows:

1. Rheumatic (inactive) heart disease	Point 1	of the 5-point q
2. Aortic insufficiency	Point 2	
3. Slight cardiac enlargement	Point 3	
4. Normal sinus rhythm		
5. Class II B	Points 4 & 5	

This indicates that: (1) There is evidence of old rheumatic infection. (2) The aortic valve has been damaged and is leaking. In this case a characteristic murmur has been heard. (3) The heart is slightly enlarged. (4) The heart's rate and rhythm are normal. (5) The patient considers himself to be moderately handicapped, II. The doctor feels that his activities should be moderately curtailed, B.

After completing this careful evaluation, it remains only for the heart specialist to transmit to the patient, and his family physician, specific recommendations regarding activity, medication and preventive measures. For everyone with definite heart disease, this type of survey should be made periodically as suggested by his physician.

Chapter III

DO YOU BELONG TO THE CORONARY CLUB?

What Is a Heart Attack?

One of the major weaknesses of our hearts has been pointed out in Chapter I. It is the necessity for heart muscle to receive its blood supply from the two small coronary arteries which arise just beyond the aortic valve. It is desirable to correlate this fact with some popular ideas about *heart attack.* This term might include any one of a number of events affecting the heart, and occurring in persons with heart disease due to a variety of causes. Actually, it appears to be applied most frequently to those who have a sudden and severe pain followed by a period of considerable disability. Such attacks are commonly the result of obstruction of a coronary artery by a clot. After such obstruction occurs there is damage to that area of heart muscle supplied by the coronary artery. Why do such clots form? Basically it is because a coronary artery has become damaged or diseased, a situation popularly known as hardening of the arteries. Such damage involves a loss of elasticity, a decrease in the

caliber of the vessel, and roughening of the otherwise smooth lining of the small artery. It includes deposits of fat and calcium in the wall of the artery. Just why obstruction occurs at a particular time is not entirely clear. It may happen after a large meal, after unusually brisk physical activity, or even after an injury such as a blow on the chest. The careful examination of many hearts at various stages after obstruction of a coronary artery show that there is a definite pattern following such an injury. Immediately after a coronary obstruction or occlusion there is damage to an area of heart muscle. Gradually, however, small twigs of adjacent coronary arteries grow into the area to take over the function of those branches that were initially deprived of blood. This capacity of the heart for repair is considerable, as you will see. The area of muscle damage is known as an *infarct*. Hence, we should have no difficulty with definitions, since the following terms refer to the same process: coronary occlusion, coronary thrombosis and cardiac infarction. We are indebted to Dr. James B. Herrick of Chicago for bringing to American physicians, early in the twentieth century, an understanding of the importance of disease of the coronary arteries. Dr. Herrick was an unusually wise but very modest physician. In the later years of his life he wrote a history of medicine, but omitted entirely to mention that he had performed this large service. Through his efforts,

and those of many other physicians in this country and abroad, we now know that a fatal attack of "acute indigestion" usually has little or nothing to do with the stomach, but is most often the result of a coronary occlusion. Having spotted the culprit, doctors have worked ceaselessly to understand this disorder better and to develop means both for preventing it and for treating it successfully.

There is the temptation to conclude that those with coronary disease face a cloudy future with progressive disability. For some this may prove to be the case. However, just as many events will take place between the certainties of birth and death, so too a member of the Coronary Club may lead a varied and productive existence subsequent to a heart attack. We have only to look about us. As illustrations we see business and professional men, many of them leaders in their fields, in spite of their membership in the Club. Even a President continued to serve, being admitted to membership while in the White House. There have been some whose memberships have been both brief and disastrous, and there are others who belong but are not aware of it. What has medical science taught us concerning the ever-growing membership of the Club?

I shall not try to present a composite picture of Club members save to say that white-collar workers appear to be in the majority. The feature ... to medical scientists is that admissions

to membership appear to be coming from men increasingly younger in age. A number of soldiers in their twenties, examined during World War II and the Korean War, were found to have this disorder. From this and other evidence we suspect that diseases of the coronary arteries may well start early in adult life, at least in the case of males. There is strong evidence from several research centers that women are relatively immune prior to the menopause, unless they happen to have diabetes or high blood pressure. This immunity of younger women is due, apparently, to a protective action related to chemicals produced by the female sex organs or ovaries. Such chemicals have been given to men with disease of their coronary arteries or hardening of arteries elsewhere. Although they tend to alleviate symptoms, they also produce undesirable changes such as enlargement of the breasts and at present are not a practical solution to the problem.

Membership in the Coronary Club is heralded by a peculiar type of pain. Often it is difficult for a patient to describe it with accuracy. It may be a pressing, squeezing, burning or bursting sensation. It usually occurs immediately beneath the breastbone, or sternum. It is particularly prone to occur if the individual attempts to walk briskly outdoors shortly after a meal. If he will stop walking it will usually subside within two or three minutes. This pain is known as *angina pectoris*. It is usually relieved by a tablet of

nitroglycerin placed under the tongue. If pain of this nature is unusually severe and persistent, is not relieved by rest or nitroglycerin and persists for fifteen minutes or longer, doctors are aware this may indicate that a branch of a coronary artery has actually become plugged by a clot. The briefer attacks of pain are thought to be due to spasm or incomplete obstruction of a coronary artery, with inadequate blood supply to heart muscle on a temporary basis. Often there is a characteristic distribution of the pain of angina pectoris. Milder attacks may center under the breastbone only. If the person continues the activity which produced the pain it is apt to spread more widely to the shoulders, along the arteries on either side of the neck, and down the inside of one or both arms.

Although patients with angina pectoris hav difficulty in describing their pain, most will agre that it is a frightening and frustrating experienc Something tells them that they must stop to re or a fatal attack may occur. This fear of dea during such pain has been well known for ce turies. Equally well established are the relatic ship to food and exertion, and the prompt bene to be secured from rest or nitroglycerin. Throu trial and error many persons learn to avoid th activities that bring on attacks. On the ot hand, there is continued need for education c cerning this important and dangerous disor During its earlier stages, particularly, and bec

it is likely to occur after meals, this type of pain may be misinterpreted as indigestion, and those suffering from coronary disease may not heed the warning in time to protect their hearts from the damage of an actual coronary occlusion. This latter event is usually sufficiently severe to require admission to a hospital or an extended period of rest in bed at home.

At this point I must interject a strong word of caution against self-diagnosis. None of us, not even a doctor, should attempt to diagnose and treat our own illness. If we have pain suspected of being angina, we should promptly consult a physician in whom we have confidence. He will examine us, as discussed in the preceding chapter, and will advise us on the basis of his well-considered opinion. Sometimes the diagnosis is difficult, since disorders of the spine, lungs, gullet, stomach, and large bowel can produce pain very similar to that of angina.

Now it can be told that we, as doctors, not infrequently see patients who go through periods of having repeated attacks of anginal pain, then are without discomfort for many years. Sometimes their pain will never return. The reasons for their recovery are not always apparent, but usually such patients have succeeded in improving their habits of living. From observation of these patients, plus an ever-increasing store of information from experiments being performed in laboratories of chemistry, biology and medi-

Adjustment of his activity to a level low enough that he does not experience pain is one of the most important things a Coronary Club patient can do to help himself. It is his major goal.

It should be emphasized that there is wide variation in the type of activities which will produce anginal pain among various members of the Club. For some, nervous tension and frustration are prime factors, to the extent that it may become necessary for them to seek a less competitive type of work than that to which they are accustomed. Such persons may find it possible to walk for considerable distances without discomfort if they are hunting, fishing, or playing golf. The same amount of walking, while under business pressure, may often produce pain.

Before their second winter as members of the Club has passed, most initiates will have learned that their level of activity, like their clothing, must vary from summer to winter. For the remainder of their lives they may find it necessary to be less active during cool or cold weather—unless they are able to spend their winter months in a warm climate.

There is also considerable variation in the responses of individuals with angina to stimulants such as tea, coffee and tobacco. Usually there will be less pain if these agents are avoided. If an episode of coronary occlusion occurs re-

quiring admission to a hospital or prolonged bed rest at home, patients can usually be persuaded to give up the use of possibly harmful drugs during the acute illness and subsequently. For those fortunate enough to have recovered from a coronary occlusion with no tendency toward angina, common-sense reduction of activity along the lines just discussed is also in order, under a doctor's guidance.

From the foregoing discussion it should be apparent that members of the Coronary Club are not urged to live a rocking-chair existence unless there are signs of severe heart failure. How is it possible to know just how active to be? The answer is not difficult, when there is a trusted physician to whom one can turn. We have already agreed that activities which produce anginal pain are to be avoided. In other respects the doctor will make suggestions based upon the findings of his examinations. His recommendations will be influenced by levels of blood pressure, the heart size, the electrocardiograph pattern and the presence or absence of symptoms and signs of heart failure. If the blood pressure is either unusually high or unusually low, if the heart is markedly enlarged, if the electrocardiogram shows unfavorable signs, or if there is evidence of congestion of the lungs or other signs of heart failure, the doctor will usually recommend very little activity. Likewise, if there is a strong

It has been demonstrated that a number of factors other than diet may increase the levels of cholesterol and other fat-like bodies in the blood. These include worry, anger, smoking, and suppression of bacterial activity in the bowel by certain antibiotics.

Through restricting sharply the amount of fa in the diet of members of the Club, attacks c pain are reduced in frequency and severity. A ready the practical applications of these and related experiments are being put to use. The observed increase in anginal attacks after a high-fat meal is thought to be due to the fact that red blood cells are crowded out by fat droplets fo a prolonged period following such meals. Thus not enough red blood cells are available for carrying oxygen to the heart muscle and other tissues until the blood is finally cleared of fat particles. The willingness to remain on a diet very low in animal and vegetable fats has, in the experience of the author and others, been the turning point toward improvement for a number of patients with angina pectoris. Two case reports are of interest in this connection.

An attorney sixty years old had suffered twc coronary occlusions within five years' time. Elec trocardiogram showed clearly that majc branches of both coronary arteries had bee obstructed. Following the second occlusion could scarcely walk as far as a city block withc having to stop because of angina. In connecti

with his examination it was discovered that he had been on a diet with liberal quantities of milk and cream to control the pain of a stomach ulcer. This has been necessary throughout most of his professional life. When he was persuaded to switch to skimmed milk and a low-fat diet, plus extra antacids for control of his ulcer distress, a remarkable change took place. His ulcer indigestion did not occur. Even more striking was the disappearance of anginal pain to the extent that he was able within three months to take a long-planned vacation in Europe. He elected to retire, and to take a trip around the world.

A businessman of fifty-five, of Pennsylvania German stock, likewise was incapacitated by angina following a coronary occlusion. When he was examined in 1958 he was unable to walk for more than one block without experiencing the familiar pressing substernal pain. His blood pressure was normal, his heart was not enlarged, and there were no evidences of heart failure. He was urged to rest for an hour after each of his meals and to try a low-fat diet (*see* list on pages 72-73). Within two weeks he was almost free from anginal pain. When he was examined a year later he was again having some pain. He had relaxed with respect to his dietary restrictions and had regained some of the weight that he had lost. He was again urged to adhere closely to his diet, particularly during cold weather.

20-GRAM FAT DIET

As suggested this diet contains approximately 72 grams of protein, 100 grams of carbohydrate, 20 grams of fat, and 900 calories. The caloric content can easily be increased by taking larger portions of the foods which contain no fat.

Your diet should include the following foods each day:

1. Foods containing fat which should be used in specified amount *only:*

Bottled skim milk or Dietene or Meritene powder made with water	1 quart
Powdered skim milk	As desired
Lean meat—beef, veal or lamb	1 small 2 oz. serving
Lean meat—chicken, liver, fish, shellfish	1 large 3-4 oz. serving

2. Foods which do not contain much fat and can be used as desired. For a nutritionally adequate diet it is advisable that you use the suggested amount:

Bread—whole grain or enriched	3 servings
Cereal—whole grain or enriched	1 serving
Fruit (one serving citrus)	3 servings
Potatoes or other starchy vegetables	2 servings
Vegetables—green leafy or yellow	1 serving
Vegetables—other	2-3 servings
Sugar	As desired
Jelly, jam, marmalade	As desired
Plain cookies, angelfood cake	As desired
Sherbet—with allowance of skim milk	As desired

Water ices	
Carbonated beverages such as ginger ale	As desired
Egg whites	As desired

3. Foods which are high in fat and should not be used at all:

   Fried foods
   Cheese other than dry cottage cheese (pot cheese), egg yolks.
   Pork, ham, bacon, sausage, luncheon meats, canned meats or fish as spam, treet, tunafish, salmon, sardines, etc.
   Whole milk, cream, ice cream, butter, oils, salad dressing, margarine
   Cakes, pies, rich cookies, hot breads, custards or puddings (unless made with skim milk and no egg yolks), chocolate, nuts (except chestnuts), or cocoanut.
   Cream soups, cream sauces or gravies

4. Suggested Menu

   Breakfast: Orange juice. Toast—2 slices with jelly
   Corn flakes, skim milk, sugar. Coffee

   Lunch: Spaghetti, tomato sauce (no meat or fat)
   String beans; bread, 2 slices with jelly.
   Fresh Pear; skim milk (tea or coffee if desired)

   Dinner: Broiled chicken (3 oz. serving)
   Rice; broccoli
   Mixed fruit salad
   Sherbet; angelfood cake
   Skim milk; coffee

Following marked improvement it is not difficult to persuade patients to adhere to a low-fat diet. It has been logical to try to find substances which will reduce blood fat levels without the need to follow a rigid low-fat diet. So far such substances have been only partially effective. Restriction of dietary fat is at present the most practical basic treatment.

There has been considerable publicity about research concerning animal versus vegetable (saturated vs. unsaturated) fats with respect to this problem. As yet the matter is not settled. Since it is better for members of the Club to be thin rather than fat, a diet limiting all fats is generally in order. With weight reduction, many patients volunteer that they haven't felt so sprightly in years. It's as though a heavy pack had been removed from their shoulders. Reduction in weight should not be too rapid, and not in excess of three pounds a week.

The exact drugs to be used in the treatment of angina will vary from patient to patient. Some will respond better to one than to another. For most it is desirable to carry small tablets of nitroglycerin, to be dissolved under the tongue as the patient stops to rest when an anginal attack occurs. Since nitroglycerin produces a fall in blood pressure, as well as improving coronary circulation, no more than two or three tablets should be taken within a period of thirty minutes, lest dizziness or fainting occur. Even

a single tablet may produce some flushing of the face and fleeting headache, but these discomforts are welcome if the anginal attack is quickly terminated as anticipated. Although even a sharp bout of pain experienced while walking will usually subside if one stops to rest for a few minutes, most Club members appreciate the prompt relief which nitroglycerin can give. Members of the Club learn early in the game that it is both painful and dangerous to keep walking once the first twinges of pain are felt.

To repeat, in the author's experience the important steps in the successful treatment of angina are a proper balance of rest and activity, avoidance of stimulants, a light, low-fat diet, and the use of nitroglycerin for control of the individual attack. Each bout of pain should also raise the question, "What have I done wrong?" "Oh yes, I hurried to the train within an hour after lunch," might be a fairly common answer.

Drugs other than nitroglycerin are available, at the discretion of your doctor, to be swallowed or inhaled. Some of these have an effective action lasting for hours, as compared with a few minutes in the case of nitroglycerin. In the author's experience these drugs are less important than fat restriction and may not be required so long as patients adhere to their diets. Nitroglycerin remains the best emergency remedy.

Sedative or tranquilizing drugs may be of especial help to those whose attacks are clearly

related to tension and frustration. Alcohol is in this category. Its effectiveness in controlling anginal pain has been known for many years. It is probably best used as a night-time sedative. A cocktail *before* dinner may increase the appetite and lead one to forget his dietary restrictions.

With respect to alcohol, the case of a traveling salesman comes to mind. Just as he was leaving New York to visit various cities across the country he experienced his first severe attack of pain under his breastbone. His first thought was to stop at a bar for a drink to relax himself. This measure produced complete but temporary relief. By taking three or four drinks a day he was able to drive to Los Angeles and back. When he reported to our clinic, his electrocardiogram showed clear evidence of recent occlusion of his left coronary artery, supplying the larger muscular half of the heart. Needless to say, he was fortunate to have survived this experience. As compared with nitroglycerin and other measures, alcohol is not a good or practical drug for controlling anginal pain.

There are other drugs which can be employed with benefit at various stages of coronary disease. The management of an acute coronary occlusion with prolonged pain will usually involve a number of medicinal agents. If the attack is of considerable severity, physicians may employ chemicals for preventing the blood from clotting, together with such drugs as oxygen and morphine.

The usefulness of anti-clotting agents has been demonstrated after a long series of ingenious experiments. Although such drugs will not dissolve a clot, once formed, they will prevent further clots from building up on the original one. The background of research required to develop these drugs is a fascinating story. Many years ago it was demonstrated that cattle were subject to a disorder known as sweet clover disease. They bled to death after eating ensilage containing sweet clover. Veterinary scientists finally isolated the chemical responsible for this bleeding tendency. It occurred to medical scientists that this same chemical might be used to prevent clotting in humans. Careful experiments were set up to test this possibility and to define safeguards necessary for the employment of this material in humans. When it was found to be safe and effective a nationwide study was set up, including many hospitals. The conditions of this experiment on humans were defined. Patients were chosen who had suffered occlusions due to clots in both arteries and veins. Of course a goodly number of these were sufferers from coronary occlusion. This nationwide study, carried on for several years, demonstrated the effectiveness of anti-clotting measures in the treatment of both venous and arterial occlusions, and has served to guide doctors throughout the world in the use of such material.

Within the past year another important ad-

quantitatively in humans. There is often definite dulling of anginal discomfort after the removal of such nerves. The operative risk involved, and the possibility of relief from more conservative types of treatment, have deterred doctors from recommending such operations frequently.

Another type of operation attempts to encourage a new blood supply to enter heart muscle. These techniques have been most ingenious, but have as yet not proved to be uniformly successful.

It has occasionally been possible to ream out sections of coronary arteries to remove both clots and arteriosclerotic debris. Such operations manifestly carry a high risk of fatality at present. Daring surgeons have even been successful, in isolated instances, in removing the weakened area of heart muscle remaining at the site of an infarct!

Although these delicate operations upon the damaged heart and its nerve supply are still in the experimental stage, they offer some promise for the future. In the process of developing them we have learned that even a badly damaged heart can be operated upon successfully. We have also learned new techniques for evaluating the coronary circulation, including means for visualizing the coronary tree, using x-rays and radio-opaque dyes.

In this chapter we have traced the natural history of coronary artery disease. Very active programs of research are being focused upon improv-

ing methods for its diagnosis and treatment, and much has been accomplished. Through application of our current knowledge, and largely as a result of proper activity and diet, the majority of patients with coronary disease can continue to live productive lives. An important advance in the treatment of acute coronary occlusion appears to be almost within our grasp, namely the possibility of actually dissolving clots in blood vessels.

Chapter IV

WHEN BLOOD PRESSURE IS TOO HIGH—HYPERTENSION

We have already mentioned the importance of taking blood pressure in the study of heart disease. When Stephen Hales succeeded in measuring the pressure within an artery of a horse he set the stage for advances in determining what pressures exist within various parts of the circulatory system. In 1896, an Italian named Riva-Rocci developed the technique of measuring blood pressure in humans with a stethoscope and sphygmomanometer much as we do it today. As a result of the work of these pioneers, supplemented by that of many others, we have come to realize the importance of the pressures which are exerted by the blood upon the walls of arteries.

High blood pressure and arteriosclerosis share the first rank among the various causes of heart disease. It is estimated that about fifteen million Americans have an increase in their blood pressure above the normal. Nearly one out of ten Americans has this problem. It is not enough to

point out the frequency of this disorder. The important thing is that marked elevation of blood pressure does produce damage to the heart and arteries when it is allowed to continue. The way in which high blood pressure produces its effect is by adding to the strain on blood vessels damaged by hardening, or arteriosclerosis. In other words, if we could have only elevation of blood pressure without associated damage to blood vessels, relatively little harm would result. In fact, this situation can be observed early in the course of high blood pressure. So long as blood vessels remain relatively healthy patients usually will not develop complications. On the other hand, progressive damage to blood vessels is prone to occur, even in younger persons, when blood pressure remains markedly elevated.

What Is Hypertension?

It is now in order to discuss the levels of blood pressure that are considered to be too high, and which warn doctors that unusual wear and tear on arteries may result. A number of reservations or conditions must be considered when we attempt to define the limits between normal blood pressure and hypertension. First of all, blood pressure is changing constantly, depending upon the emotional state and the type of physical activity. Even while resting many persons will show phasic variations which illustrate the dynamic nature of the nerve reflexes controlling the cir-

culation, as discussed in Chapter I. Most of us will show some alteration in blood pressure regardless of activity, and related to emotional changes or to noises arising adjacent to the examining room. Hence the use of blood pressure as a part of the lie detector test. Patients with an anxious temperament may show fluctuations in systolic pressures of from twenty to thirty points within a few seconds. For these reasons your doctor may decline to reveal to you your exact pressure, as does the author with his patients. The lower or diastolic pressure is not apt to fluctuate as widely as the systolic, but it is a more important clue to impending danger than is the systolic. There is evidence that a person is in more danger when his blood pressure remains constantly elevated than when there is fluctuation between normal and high pressure.

The figures used to designate blood pressure refer to a column of mercury as the standard. Systolic pressure may vary normally between 90 and 150 millimeters of mercury. Diastolic pressure normally ranges between 65 and 90. These are expressed as 90/65 and 150/90. There are accepted variations in blood pressure related to age and to other factors. The old adage that one's (systolic) blood pressure should be 100 plus his age is in general borne out. In other words, as we grow older our systolic pressures tend to rise gradually, and more markedly than do the diastolic pressures.

What Makes Blood Pressure?

Blood pressure, as has been pointed out, is a part of the intricate balance which the body maintains through reflex control. Given a situation which produces either a rise or fall in pressure, these reflexes act with lightning rapidity to tend to restore the balance.

It is now in order to define some of the factors of greatest importance in the maintenance of our blood pressure. There are five of these: The *pumping action* of the heart is the basic factor producing blood pressure. We have already seen that pressures measured in the arm or elsewhere reflect the maximal and minimal pressure associated with each heartbeat. Drugs that increase the strength of the heartbeat are prone to increase levels of blood pressure, despite the reflex controls which are constantly operating to prevent changes in pressure. Contrarily, when the pump is weak, as after extensive myocardial infarction (damage to heart muscle), both systolic and diastolic blood pressures are prone to be low. In this circumstance, the systolic blood pressure is frequently disproportionally low, such as 120/100. As the heart muscle regains its strength during convalescence from a coronary occlusion, the systolic pressure will rise more markedly than the diastolic, with resulting pressures in the range of 160/100, as compared with the lower

igure recorded at the height of injury to heart nuscle.

Second only in importance to the heart pump or maintaining blood pressure is the *resistance* ›ffered *by the walls of the arteries,* including heir smaller branches. This is easily illustrated ›y adjusting the nozzle of a garden hose. As the tream of water is made smaller, one can feel ncreased pressure within the hose, and the tream of water leaving the hose is smaller, onger and under increased pressure. In the conlition known as shock or circulatory collapse, when the reflex control of blood vessel resistance is no longer effective, blood pressure will fall to dangerously low levels and the pulse may disappear. Doctors have learned that the vital organs of the body suffer severe damage during such shock. The emergency treatment of shock introduces the third factor for the maintenance of normal blood pressure.

The *quantity* of blood in the arterial system manifestly has an important bearing on the level of blood pressure. To put it tersely: no blood, no blood pressure. In shock, because of the relaxation of the blood vessels, there is a relative lack of circulating blood. Accordingly, a transfusion of blood or plasma is usually started as one of the first measures to combat shock. At the same time drugs can be given to increase the tone, or resistance, of the blood vessels, thus al-

each ventricular contraction. When this valve is destroyed or rendered incompetent the diastolic pressure may be quite low, sometimes even 0.

It is not difficult to find examples to illustrate how blood pressure has become too high or has fallen too low because of disturbances in one or more of the factors above. More often, the body's adaptability will allow blood pressures to remain relatively stable in spite of major disturbances: A woman of seventy was under observation because her blood pressure was elevated in the range of 200/110. This pressure reflected a loss of elasticity due to arteriosclerosis. During her later life she suffered two strokes and a coronary occlusion. It also became evident that her aorta was considerably dilated, due to the combination of hypertension and arteriosclerosis. This enlargement of her chief artery could be seen with a fluoroscope to involve the entire aorta in its course within the chest. Furthermore, similar dilatation could easily be felt in the abdomen. During her final illness, she began to have pain about her right hip, which suggested that there might be leakage of blood from the abdominal aorta, giving rise to pressure on nerves about her hip. Accordingly she was admitted to the hospital. Although she was in considerable discomfort, her blood pressures remained markedly elevated. It then became clearly evident that leakage was occurring. The reflex control of her blood pressure would momentarily fail, with resulting sharp but

transient declines in both systolic and diastolic pressures. It was felt that she would not withstand a major abdominal operation in an attempt to repair the leak. Finally her blood pressure fell persistently and she died shortly thereafter. Within her abdominal cavity was found more than a quart of blood. That her pressure remained fairly constant until very close to the end of her life was an example of the body's adjustment to blood loss.

THE CAUSES OF HIGH BLOOD PRESSURE

From the foregoing discussion of factors concerned in maintaining blood pressure it would be easy to conclude that a disturbance of any one of five mechanisms involved might result in persistent hypertension. Actually, the problem is not quite so complex in some ways. It has been possible, as the result of continued research, to test the importance in animals and humans of these various factors. Experiments have been designed to keep four of the factors relatively constant while changing a fifth, and so to estimate the relative importance of each. Although some examples have been cited of how these factors can influence blood pressure in humans, most of the evidence points toward the fact that an *increase in the resistance* of the smaller arteries (*arterioles*) is responsible for the persistent elevation of blood pressure seen in most patients with hypertension. This conclusion has been reached

following numerous studies in both humans and animals, involving the use of elaborate instruments. In humans, testing of the other four factors has resulted in their virtual exclusion. Accordingly, it has been possible to focus attention on those things which might explain the increase in resistance observed in human hypertension.

In association with the increased resistance of the arterioles in hypertension, the nozzle of the hose squirts a smaller jet. The problem has been, therefore, to discover why the jet is smaller, or why the arterioles are contracted: Such contraction might be the result of prolonged bombardment by nervous stimuli, actual swelling of the walls of the arterioles making them thicker than normal, or to chemical substances producing contraction of these vessels.

Do Nerves Do It?

It has been amply demonstrated that the nervous system can exert important effects upon blood pressure, as illustrated by the use of the lie detector. Persons with high blood pressure may appear to have a nervous temperament. It would be easy to conclude that hypertension is simply due to nervousness. Furthermore, in the experimental animal one can produce high blood pressure through stimulation of the brain by increasing the pressure within the skull. Another type of "nervous" hypertension will result from cutting those nerves in the neck or chest which act

to salt their food freely. Accordingly doctors have long advised patients to avoid salt if their blood pressures are too high. Through very careful studies involving the salt content of body tissues and fluids we have come to the realization that the contraction of arterioles may well be due to some as yet unexplained change on their part which makes them retain abnormal amounts of salt. According to chemical laws, this tendency is followed by inpouring of fluid. The result may be swelling of arteriolar walls. Subsequently we shall discuss the practical applications of this possibility with respect to the treatment of high blood pressure.

Another possibility for explaining the contraction of arteriolar walls is that some chemical may produce a prolonged contraction, just as though nerve stimulation were long continued. During the past twenty years many experiments, carefully designed, have been conducted to study the possibility. Various types of chemicals have been suspected. It has long been known that the pituitary gland, situated under the base of the brain, secretes into the blood stream a substance (*pituitrin*) which has the ability to produce such a constriction of arterioles. Injections of pituitrin will indeed produce a rise in blood pressure. The adrenal glands, which lie immediately above either kidney, have been shown to produce a substance (*adrenalin*) which has a similar action. Furthermore, another part of the adrenal gland

produces various substances related to *cortisone* which exert a powerful effect upon the retention of salt in the body. It has been natural for scientists to explore the possibility that secretions of the pituitary or adrenals might be the cause of hypertension in man. It has been learned that tumors of the pituitary and adrenal glands can indeed produce hypertension that can be cured by their removal. On the other hand, we have yet to find convincing evidence that these glands, in the absence of tumors, can be responsible for hypertension as seen in humans.

While exploring adrenal and pituitary factors, scientists have also busily sought for chemicals produced elsewhere which might account for the increased arteriolar contraction in hypertension. The problem resolved itself at one point to a very straightforward matter: Could we produce in an experimental animal hypertension which was similar to or identical with hypertension as seen in the majority of humans? We have already seen that the so-called nerve types of hypertension do not fulfill this requirement. A major breakthrough occurred in about 1934, following a series of very careful and ingenious experiments performed by Dr. Harry Goldblatt of Cleveland. Using a colony of healthy dogs, he was able to produce hypertension through the application of small silver clamps to the main arteries supplying their kidneys. In fact, such a clamp properly placed on the artery of one kid-

ney would produce the disorder, which could be cured subsequently by removing the affected kidney. Like human hypertension, the high blood pressure so produced in dogs was associated with an increased arteriolar resistance as its chief and salient characteristic. Furthermore, such experiments coincided with experience in humans to suggest that the kidney was frequently at fault in the presence of hypertension. In animals and in man it has become apparent that disorders of the kidney, including Bright's disease (*nephritis*), infections, obstructions to urinary flow, tumors and kidney stones all can produce elevations in blood pressure. Through Dr. Goldblatt's method we were able to prepare at will an example of high blood pressure which corresponded very closely with human hypertension, and which allowed us to study what types of chemicals might be responsible for the rise in blood pressure.

To make a long story of elaborate experimentation short, our present concept is as follows: Impairment of the arterial supply of the kidney of dogs (and probably of humans) results in the production of an abnormal substance, *renin*, by the affected kidney. This reacts with other chemicals produced in the liver to form a substance, *angiotensin* which is capable of producing a sharp rise in blood pressure in association with arteriolar contraction. We are now at the somewhat frustrating stage that renin can be demonstrated occasionally in patients with high blood

pressure. On the other hand angiotensin has not yet been recovered from humans, presumably because it is very quickly destroyed in connection with its action on the arteriolar walls. We have gone further, however, to synthesize angiotensin. This has allowed us to study its actions more closely, and to search for chemicals which might counteract its blood-pressure-raising qualities.

In the foregoing discussion of the parts played by the arterioles and kidneys in the production of high blood pressure, considerable simplification has been employed. It remains a distinct possibility that, although the kidney may be the first step, the pituitary and adrenal may well enter into the maintenance of high blood pressure. These possibilities will be further discussed in the consideration of present-day treatment of hypertension.

The Patient with Hypertension

A review of the various factors which may cause hypertension indicates that persistent elevation of blood pressure can be due to a variety of abnormalities. When a patient is discovered to have hypertension he is subjected to a searching examination similar to that employed in those suspected of having damaged coronary arteries. The doctor is again concerned to know the time and nature of first symptoms, and what has been noted subsequently. Since various diseases of the kidneys can give rise to hyperten-

sion, a background of scarlet fever or repeated sore throat in childhood, or of kidney stone or infection later, may be valuable clues to guide the selection of special tests. As before, the total picture is worked out, and the Five-Point Quiz is again applied. In addition, during his study of patients with high blood pressure, the doctor will seek the answer to five questions which are particularly pertinent to patients with hypertension: (1) What cause or causes for hypertension are evident? (2) How marked is the elevation of pressure? (3) Has any damage been done to the small arteries in the eye, brain, heart and kidneys? (4) Is any general disorder present, such as diabetes, obesity, or disturbances of thyroid function? (5) What treatment is in order?

At this point an aside is in order. Although it is necessary, for the sake of clarity and simplicity, to consider coronary artery disease and hypertension separately, they occur together frequently. Indeed, the combination of hypertension in association with arterial damage (*arteriosclerosis*) is many times more frequent than the occurrence of either disorder by itself. Again it should be evident that the complication of hypertension most to be feared is advancing arteriosclerosis. Sometimes this cycle is found: If there is arteriosclerotic obstruction of a kidney artery, a marked elevation of blood pressure can result, which under favorable circumstances can be corrected when the arterial obstruction is relieved, or the

here. Let us therefore proceed to a consideration of how the doctor interprets the variations of blood pressures in the individual case. We have already seen that an individual's pressure may vary considerably, in response to emotional or other causes. If a man shows blood pressure of 140/80 and 200/120 on two examinations, which is correct? The answer is "neither." For the correct answer the doctor must have a sufficient number of readings, over a period of days or weeks, and taken when the patient is as relaxed as possible. He may wish to keep a record in relation to the day's worries or activities. Although it might be desirable also to have a record of pressure obtained at the height of the individual's most vigorous activities, this is not feasible. Well-designed experiments confirm that this pressure, particularly in hypertensive individuals, may be alarmingly high. It has been found that most individuals will have a fairly constant range, if pressure is measured after a short period of rest in as relaxing an atmosphere as possible. Some doctors even train members of the patient's family to take blood pressure in the quiet environment of their homes, to supplement office readings. Since some of the most powerful drugs for reducing pressure exert their maximal effects when the patient is in the upright position, doctors may take blood pressures in both lying and standing positions.

Blood pressure alone is of little value in plan-

ning treatment or assessing the severity of hypertensive heart disease. For example, I can recall a patient who visited our clinic regularly over a period of ten years beginning about 1940. Throughout this period of time her systolic blood pressure, when measured in the clinic, was never under 300! Her diastolic pressure, however, was never more than 100. She illustrated the fact that women are apt to tolerate hypertension better than men, and that diastolic pressure is more critical than systolic. Had her diastolic pressure been over 120, at a time when effective treatment was not available, she would have been fortunate to have survived for ten years. As we examined her repeatedly it was apparent that, despite her high systolic pressure, various organs in her body did not show evidence of the progressive deterioration which would probably have occurred if her diastolic levels also had been markedly elevated. It was the over-all picture that allowed us to observe her course without alarm.

How does the doctor explore the amount of damage suffered by various organs in the presence of hypertension? Again, this information is obtained from a variety of sources in order to obtain proper perspective. The patient's symptoms offer useful leads. Morning headaches, dizziness, or fleeting numbness and weakness of a hand or leg suggest impaired blood supply to the brain. Angina of effort, or cramps in the calves of the legs upon walking, indicate damage

to the coronary or leg arteries. We have already pointed out that such symptoms as shortness of breath and puffiness of the ankles may be evidences of heart failure.

Using these symptoms as a guide, the doctor proceeds with his physical examination in the same methodical fashion as for the evaluation of suspected coronary disease. He is apt to be particularly interested in the eyes, and may request a consultation with an *ophthalmologist*, a physician eye specialist. For it is only in the back of the eyes that we can actually see "naked" *arterioles*. In other areas we can at best only see pulsations or feel larger arteries, or obtain other indirect evidence of arterial damage. As before, the size of the heart, the presence or absence of abnormalities heard with the stethoscope, and the electrocardiogram, are the standard approach with respect to damage to the heart, as evaluated in connection with the patient's symptoms. Other more elaborate tests can be applied if necessary. Symptoms suggesting heart failure may or may not be accompanied by confirmatory signs upon physical examination. Again it is a matter of correlation to determine the true state of affairs.

Special tests to probe for possible causes of the elevation of blood pressure often do double duty. For example, in addition to showing abnormalities of the kidney which can be the cause of hypertension, they may also give evidence of the amount of damage which these organs have suf-

fered. Thus, when his patient has completed the diagnostic survey (which may require several days in the hospital), the doctor may draw conclusions such as the following.

"Although Mrs. Smith's blood pressure averages 200/100, she shows no evidence of blood vessel damage in the brain, eyes, heart, or kidneys. She is seventy years old and feels quite well. Her habits of living are good, and in keeping with her age. She will be urged to rest for an hour after each of her meals, avoid stair-climbing and restrict her salt and fat intake moderately. Drugs will be given to promote relaxation and to avoid retention of salt."

"Mr. Jones's blood pressure averages 180/100. Considerable arteriosclerosis is visable in his eyes, together with three small hemorrhages. His heart is not enlarged, and his electrocardiogram remains normal. On the other hand, he has involvement of his coronary arteries, with a clear history of angina pectoris during the past two years. He is overweight and requires carefu treatment."

"Mr. Thompson was not aware until si months ago that his blood pressure was abno mal. However, his last physical examination w four years ago. Although his pressure averag only 170/90, kidney and blood tests show ve

marked impairment of his kidney function. As a background he gives a history of passing a kidney stone twelve years ago. Now his margin of safety is very slender. Efforts will be made to correct a smoldering infection of his kidneys, but it is too late to hope to improve kidney function."

Treatment. The Conquest of High Blood Pressure

The conquest of high blood pressure includes several triumphs of medical research. For many years it has been recognized that sodium chloride (table salt) has played a part in the origin and persistence of at least certain types of high blood pressure. For instance, some persons with a tendency toward hypertension will show a further rise in pressure if solutions of salt are injected into their veins. Those with normal pressure can receive the same injections without showing a rise in blood pressure. From such relatively simple observations as these the pursuit of the salt factor has led investigators into complex studies, demanding intricate instruments, and with expenditures for research totaling millions of dollars over the years.

It was necessary to study, by experiments slanted toward various ends, the organs known to be concerned with the disposition of salt and water in the body. These included the kidneys, heart, and the pituitary and adrenal glands. At

times the trail was hard to follow, but the objective remained clear as a snow-capped mountain peak: Why is salt so very important in the hypertension problem? Sometimes an earnest worker or team would seem to accomplish in a few years what others had been unable to achieve in decades. The discovery by Dr. Goldblatt of the importance of disturbances in the kidney's arterial supply was a major breakthrough. Likewise it was found that some persons almost blind or dead from hypertension would show striking improvement so long as they ate a diet containing only rice, fruit, and sugar. The chief merit of this diet has been demonstrated to be its low content of salt.

The role of the adrenal glands in the maintenance of blood pressure has already been mentioned. It is now in order to point out that these two little glands, lying above each kidney, are of vital importance. If they are damaged by disease, their owners will show low blood pressure and will tend to lose abnormal amounts of salt. This disease, first described in 1855 by an English physician, Thomas Addison, has come to bear his name. In the decades that followed, many careful investigations of adrenal function have been performed, showing that the adrenals are intimately concerned with the disposition of salt and water. At one stage, a medical feud arose between two groups of investigators. One held that the chief function of the outer region (*cor-*

tex) of the adrenal gland had as its chief function the control of salt and water in the body. The other camp stoutly upheld that an even more important function of the cortex was in the disposition of sugar and its chemical relatives. Now we know that all these, and other functions, are within the province of the adrenal cortex, in intimate association with other glands of the body, including the pituitary, thyroid, and pancreas. This brief glance backward will, I hope, serve to suggest how medical research can advance by gradual stages, each investigator standing on the shoulders of those who have previously struggled with the same or related problems.

Having this body of information indicating that the adrenal cortex plays an important part in regulating blood pressure and the distribution of salt within the body it was natural that doctors should try the effect of removing all or a part of the adrenal glands from patients having high blood pressure. Early attempts were not successful: If too little adrenal tissue was removed, no effect upon blood pressure was observed. Contrarily, if too much was taken out, patients died for lack of the secretion of these vital organs.

By 1950 the life-giving hormone of the adrenal cortex (*cortisone*) had been discovered, and was being produced for the treatment of such disorders as rheumatism and Addison's disease. Doctors embarked upon a careful study of the effects

times the trail was hard to follow, but the objective remained clear as a snow-capped mountain peak: Why is salt so very important in the hypertension problem? Sometimes an earnest worker or team would seem to accomplish in a few years what others had been unable to achieve in decades. The discovery by Dr. Goldblatt of the importance of disturbances in the kidney's arterial supply was a major breakthrough. Likewise it was found that some persons almost blind or dead from hypertension would show striking improvement so long as they ate a diet containing only rice, fruit, and sugar. The chief merit of this diet has been demonstrated to be its low content of salt.

The role of the adrenal glands in the maintenance of blood pressure has already been mentioned. It is now in order to point out that these two little glands, lying above each kidney, are of vital importance. If they are damaged by disease, their owners will show low blood pressure and will tend to lose abnormal amounts of salt. This disease, first described in 1855 by an English physician, Thomas Addison, has come to bear his name. In the decades that followed, many careful investigations of adrenal function have been performed, showing that the adrenals are intimately concerned with the disposition of salt and water. At one stage, a medical feud arose between two groups of investigators. One held that the chief function of the outer region (cor-

agents, such as the tranquilizers, with the gradual addition of the more potent ones as necessary. Careful medical supervision must be maintained, with periodic re-evaluation to assess possible damage to arteries throughout the body. Since arteriosclerosis exists hand in hand with hypertension, treatment will be designed to combat both disorders.

The recent discovery of highly effective measures for controlling hypertension deserves a word of caution, and another of hope. Doctors must not be lulled into a sense of false security just because we can reduce high levels of blood pressure. We dare not relax constant vigilance in our attempts to answer medicine's most urgent question, "What is the cause?"

The ray of hope is this: A small proportion of patients with *severe hypertension* may, after months of successful medical treatment, revert to a fortunate state wherein blood pressure remains normal with little or no medication. Perhaps this minority will enlarge as our knowledge and experience increase.

Chapter V

BOTH VALVES AND MUSCLE: RHEUMATIC HEART DISEASE

The Nature of Rheumatic Fever

We have already mentioned heart murmurs; some are insignificant, but some reflect abnormalities. Formerly doctors had to judge the importance of murmurs, as heard through a stethoscope, upon the basis of their location, intensity and character, together with changes reflected in the heart and other parts of the body. With the development, through research, of methods for placing catheters within the heart and blood vessels, and the use of x-ray techniques allowing these structures to be visualized, it has become possible to evaluate heart murmurs more precisely. Not only can we identify the origin of a murmur, but we can obtain evidence concerning the effects of the underlying abnormality upon the heart and the circulation. Deformities or enlargement of various chambers of the heart can be visualized, as can obstruction, narrowing, or of dilatation of veins or arteries. Exact pressure

and content of oxygen and carbon dioxide can be measured as the tip of a catheter is placed accurately under guidance by x-ray. Through techniques such as these research has allowed doctors to enhance tremendously their ability to interpret heart murmurs, as well as to understand other abnormalities of the heart and circulation.

The disease which commonly produces deformities of heart valves is rheumatic fever, a disorder more common in areas where the winters are cold and wet. Many of you are aware of the relationship between infection by a special type of germ, the *streptococcus*, and rheumatic fever. Germs of this type may also cause the related infections scarlet fever, acute sore throat, and inflammation of the kidneys (*nephritis*).

The valvular deformity of rheumatic fever is not due to actual *infection* in the heart valves, but is the result of an allergic response to a streptococcic infection in the throat or elsewhere: Just as the allergic skin eruptions of hives in a person sensitive to chocolate do not contain particles of chocolate, neither do the inflamed valves in rheumatic fever contain streptococci. During the height of the attack the heart muscle as well as the valves may become inflamed. Furthermore, the tissue around the heart (*pericardium*) is often involved. Is it any wonder that heart failure can be the result, and that children suffering from acute rheumatic fever must be kept

at rest until all evidence of active infection has subsided?

Rheumatic fever is a stealthy disease. It may occur as a smoldering fire which is difficult to detect; it may burst into flame again after it has apparently been extinguished. Not uncommonly adults will give clear evidence of rheumatic valvular disease without having suffered a typical attack of rheumatic fever in childhood.

There is also the unfortunate tendency for heart valves which have become deformed during an attack of rheumatic fever to become *infected* by the *streptococcus* or other germs later in life. Often it can be demonstrated that these late infections have arisen from infections about the roots of the teeth. For this reason both children and adults who have suffered an attack of rheumatic fever should have careful examinations of their teeth twice yearly for the rest of their lives. It is the purpose of this chapter to show how advances in medical research have allowed us to prevent in large part the ravages of this disease, or to compensate by medical and surgical treatment for the damage done.

Prevention and Treatment of the Acute Attack

Careful studies performed upon children and adults during seasons when colds are prevalent have shown that the use of penicillin and other *antibiotics* will, by ridding the throat of strep-

tococci, tend to prevent the occurrence of acute rheumatic fever as a complication of tonsillitis and sore throat. Such experience has pointed the way toward effective prevention of rheumatic fever. Furthermore, the use of antibiotics throughout the year will often protect from subsequent activation patients who have actually suffered an attack of rheumatic fever.

Antibiotics also have a place in the treatment of acute attacks of rheumatic fever, if streptococci are found in cultures from the throat. Aspirin and related drugs have long been relied upon in the treatment of acute rheumatic fever, as in chronic rheumatism. But something new has been added in the form of hormones of the adrenal cortex (*cortisone*), and a secretion of the pituitary gland (ACTH) which acts by stimulating the adrenal to increase its output of cortisone. (Yes, this is the same cortisone that is used following adrenalectomy operations for hypertension!)

The case of David R. will illustrate how cortisone may be lifesaving. He is now a sophomore in college. His pediatrician asked me to see him one winter night in 1950, when David was only nine years old. He had been admitted to the hospital with a fever of 105, blue, and gasping for breath. Through the stethoscope a crunching sound of pericardial inflammation was heard over his markedly enlarged heart. We concurred in a tentative diagnosis of acute rheumatic fever,

and agreed that he should have both an antibiotic and cortisone. By the next morning David had only a slight fever, was breathing comfortably, and looked like a different boy. His pediatrician subsequently reduced the doses of antibiotics and cortisone; David has required neither during the past two years. By being tutored at home during the first winter he was able to keep up with his class at school. It has been gratifying to examine him twice annually over the ensuing years. His heart, originally enlarged, became normal during his initial hospitalization. Shortly after the lifesaving effects of cortisone and antibiotics had first become evident in his case, his pediatrician pointed out that David appeared to have a disorder related to, but not actually acute rheumatic fever. Sure enough, some months later he developed persistent swellings of his fingers and wrists which required the temporary use of larger doses of cortisone. The persistent swelling of both hands, and the freedom of his heart valves from inflammation, were not in keeping with a diagnosis of acute rheumatic fever. It b came apparent that the diagnosis was Still's di ease, or juvenile rheumatoid arthritis. His ca is presented because it illustrates what can achieved through the use of these new drugs those critically ill with rheumatic fever and lated disorders. It also shows the importance careful treatment for long periods of time lowing initial success.

And Further Care

As illustrated by the case of David, a child who has had rheumatic fever should have careful follow-up examinations by his doctor. The objectives of such care are to prevent further attacks, and to meet such complications as may arise. If the patient should catch cold, the doctor should be notified. He should visit his dentist at least twice annually, to allow correction of cavities which might harbor bacterial infection. At times special blood tests will be required to determine whether or not smoldering rheumatic activity is present.

It is expected that the use of such new agents as antibiotics and cortisone will mean that fewer children will have valvular damage and enlarged hearts following rheumatic fever. Sufficient time has not yet passed since the discovery of these aids to know just how great the gain in health will be. Meanwhile, discoveries in the realm of surgery have made it possible to help many who would otherwise have been consigned to lives of chronic heart failure and inactivity.

Types of Valvular Deformity

The deformities of the heart's valves due to acute rheumatic fever are of two types: (1) The valve leaflets may become permanently separated, so that leakage occurs; or (2) they may become fused together so that the opening is not large

enough to allow a proper flow of blood during the contraction of the corresponding auricle or ventricle. Not infrequently a single valve will show both types of deformity. In any event the mechanics of the heart may be seriously disturbed and heart failure may eventually result. Precise methods have been devised for measuring the degree of disturbances associated with damage to the heart's valves. As described previously, pressure can be measured within all four chambers of the heart, and the chambers can be visualized by injecting material opaque to x-rays Utilizing these techniques, it is possible not only to detect important effects of valvular heart disease, but also to determine when the degree of strain on the heart and lungs is sufficiently severe to warrant operation.

Surgical Treatment

The achievements of modern surgery with respect to operations upon the heart, and the promise of even more exciting advances in the future, deserve some explanation. Surgery of the heart was long waiting to be born. The anatomy or structure of both the heart and its surrounding organs has been well known for centuries. It was no secret, for instance, what structures lay adjacent to the mitral valve, to be encountered by the surgeon who might wish to approach this region—a commonplace operation today. The steps which had to be taken to achieve this

modern miracle have been carried out within little more than the past century.

The conquest of pain during operation marks the beginning of modern surgery. So long as pain and shock were accompaniments of all operations, large or small, surgery could not advance. With the discovery, in 1846, that general anesthesia was feasible the first step was taken.

To Louis Pasteur we must give credit for his gift to humanity which initiated the second step: Pasteur demonstrated that infection is caused by bacteria. Subsequently it was necessary to determine how best to prevent infection—by avoiding bacterial contamination through cleanliness, or by sterilization with heat or chemical agents. In time to prevent the loss of countless lives during World War II, penicillin was discovered by Sir Alexander Fleming. Its discovery was the by-product of a chance observation in the laboratory. To produce it in sufficient quantity commercially was quite another matter, for which Dr. A. N. Richards of Philadelphia and others deserve great credit. Penicillin, and the growing family of antibiotics, are substances which inhibit or stop bacterial growth. They are given to patients by injection or by mouth, are absorbed into the blood stream, and hence are distributed throughout the body.

In 1883, the Viennese surgeon Billroth pronounced, "The surgeon who should attempt to suture [sew up] a wound of the heart would

lose the respect of his colleagues." Only thirteen years later Rehn reported the first successful suture of a heart wound. Advances in the understanding of the functions of the heart and lungs, and the importance of replacing the blood lost at operation, followed in steady progression. In 1933, Dr. Evarts Graham of St. Louis first successfully removed an entire lung. By the end of the Second World War the stage was set for successful operations upon and within the heart. Now at last we are in the process of developing machines for taking over the function of the heart and lungs. With them the heart can be stopped, opened, the defect repaired, and then the heartbeat restarted. Improvements in the types and uses of anesthetics has played a large part in our ability to perform these modern miracles. We have learned that if the body's temperature is reduced (as with ice packs) it will better tolerate interruption of the circulation while critical operations are being performed.

A brief review, further, of some of the surgical "firsts" relating to the heart will serve to show how recently these achievements have occurred. In 1939, Dr. Robert E. Gross of Boston succeeded in operating successfully upon a patent ductus arteriosus. This condition is a holdover from prenatal life. It is a communication (outside the heart) between the aorta and the pulmonary

artery. If allowed to persist into puberty or adult life, heart failure may occur.

Doctors C. Crafoord and G. Nylin, in Sweden, succeeded, in 1944, in an attempt to remove the constricted portion of the aorta producing another congenital defect known as *coarctation.*

In 1945, Doctors Alfred Blalock and Helen B. Taussig of Baltimore devised the first successful operation for the tetralogy of Fallot, a combination of congenital defects which is a frequent cause of blue babies.

In 1949, Doctors Charles P. Bailey, Robert P. Glover, and T. J. O'Neill of Philadelphia reported their successful correction of rheumatic mitral stenosis (narrowing of the rheumatic orifice). A single such operation had been performed successfully by Dr. Soutter in England in 1925.

Subsequently operations have been devised for correction of deformities, congenital or rheumatic, involving the three valves other than the mitral, for closing abnormal openings between the right and the left sides of the heart, and for improving congenital abnormalities of the arteries and veins about the heart. Following years of intensive research, Dr. John H. Gibbon of Philadelphia, in 1954, used the pump oxygenator (heart-lung machine), which he had developed, for the first successful repair of a congenital interatrial septal defect in a child. In each case,

through refinements in diagnostic methods, research has allowed us to identify these abnormalities with certainty prior to operation.

A word of caution here is in order. When we take our automobiles to a garage for repairs we often receive estimates in advance concerning the nature and cost of the work required. If the difficulty is not corrected we may decide to trade for a new model. By contrast, surgery of the heart valves is usually for keeps. A slight miscalculation or technical fault may mean a dead patient. A second operation usually means a larger risk than the first. It is for these reasons that we apply the newer surgical advances with the utmost circumspection. No single physician or surgeon can assume the whole responsibility. The recommendation to operate is reached after a careful review of the patient's past course, his response to various types of medicines, the exact nature of his valvular deformity, his likely future without surgery, and the known risks of the operation planned.

At the time of operation the highly skilled chest and heart surgeon is aided by a supporting cast of experts in nursing, anesthesiology, medicine and chemistry. It is the surgeon's responsibility to decide at the operating table whether or not he can safely complete the operation planned. A view of the operating room, in action, will be presented in Chapter VI. The rapid exchange of medical information among

doctors allows us to give you and your family the benefit of researches performed in all parts of the world.

One of the greatest contributions of modern training is the concept that surgery does not begin or end in the operating room. It begins with the careful evaluation of the patient prior to operation, using all available information, and continues throughout the years in medical and surgical follow-up examinations. Of course the operation itself is critical. But immediately after successful operations patients will succumb needlessly unless they are watched day and night by a well-coordinated team of doctors and nurses.

Your reward, and ours as physicians and surgeons, is the happy experience of seeing those who seemed doomed to lives of discomfort and inactivity restored to near-normal health. In this connection, the case of Mrs. N. comes to mind. She had come from a considerable distance, sent by her family physician to determine whether or not an operation on her heart might be performed with safety and benefit. An attack of rheumatic fever in childhood had left her with clear evidence of narrowing of the mitral valve (between the left atrium and the left ventricle), with a characteristic murmur. Yet her heart was not enlarged, and her ability to perform various activities was excellent. In addition to doing all her own housework for her family of four, she

currently effective, but hazardous operations upon heart valves are to be employed only after treatment with medicines has been given a thorough trial, and after special tests have been employed to predict the necessity for operation and the probable degree of success.

We have seen in the case of David that rheumatic fever is one of a family of related diseases. The success with which it is now possible to *diagnose* or identify the individuals in this family means that we can also *treat* each more effectively. This success has followed many careful experiments performed separately or as a part of the observation of patients with the disorders, and correlated with laboratory work in chemistry, bacteriology, and the related sciences.

From the foregoing it is apparent that much progress is being made in the treatment of rheumatic heart disease. Although the surgical correction of valvular deformities is important and dramatic, the medical treatment of rheumatic fever has even wider usefulness, and the prevention of rheumatic fever is basic. In the battles for prevention and early treatment, doctors are aided by an army of nurses, teachers, social workers, and other skilled personnel, whose influence pervades the communities in which they work. Together with the doctors, they bring hope and help to those who know and fear the dangers of rheumatic fever.

Chapter VI

BLUE BABIES OR PINK: CONGENITAL HEART DISEASE

During their growth for nine months prior to birth, babies pass through a series of complex changes, beginning with a single cell, then advance to stages resembling progressively more complex lower animals until at last they are ready to be born as little pocket editions of men and women.

The heart and blood vessels share in this remarkable series of transformations. Starting with a simple tube like the heart of an earthworm, the human heart rotates upon itself and utilizes various of the primitive blood vessels to attain the nearly adult stage required by a newborn infant.

Prior to birth, when no air enters the lungs, the infant receives oxygen from its mother's circulation. During this time a thin-walled vessel, the *ductus arteriosus*, conducts blood directly from the aorta to the pulmonary artery (see Diagram 1) and acts as a short-circuit or shunt which virtually prevents the lungs from receiving

blood. Another shunt, the *foramen ovale* exists as an opening between the two atria prior to birth. As the newborn infant takes his first gasps of air, filling his lungs, these two shunts begin to close. Within weeks after birth these channels are obliterated, thus completing the separation of the lung from the body circulation, as is the case in all humans and higher animals.

The Hand of the Potter Can Shake

With this intricate series of changes occurring not only during the early stages of pregnancy, but also at the time of birth, is it any wonder that slight or marked imperfections can occur? Such deviations from normal comprise *congenital* (*with-birth*) heart disease. It is the purpose of this chapter to show that the proportionately small, but precious group of children having congenital abnormalities (*anomalies*) of the heart and circulation can now have the benefit of accurate diagnosis, and in most instances can be helped by modern surgical treatment. Some advances in the development of operations within the heart have already been cited in Chapter V. As suggested earlier, we are even beginning to have some appreciation of factors which may play a part in interfering with the orderly development of the heart and other organs prior to birth.

Congenital abnormalities of the heart and

circulation can be divided into: (1) Those occurring within or outside the heart; (2) those producing no impairment of oxygenation of the blood (*acyanotic*); and (3) those predisposing to poor oxygenation (*cyanosis*). To this last group belong the blue babies.

From the standpoint of surgical correction those congenital abnormalities lying outside the heart are in general more easily corrected. Not infrequently, defects within the heart may be present at the same time. In order that precise corrections can be made during operation it is necessary that the exact nature of defects both inside and outside the heart be known prior to operation. It is here that advances in experimental medicine and surgery have allowed us to reap a satisfying reward.

The Diagnosis of Congenital Heart Disease

Only twenty years ago the diagnosis of congenital heart disease was based upon the appearance and symptoms of the patient, blood pressure in the arms and legs, and the character of murmurs heard over the heart and great vessels. At best the diagnosis, from this information, was a shrewd guess and subject to considerable error. Through the adaptation of techniques learned in animal experimentation it was discovered that the precise nature of congenital anomalies could be determined. Small

hollow tubes (catheters) have been placed within the heart and blood vessels, producing little or no discomfort to the patient, and allowing doctors to determine pressures and content of oxygen. In addition, the size and shape of blood vessels and of the individual chambers of the heart can be visualized just after the injection of chemicals opaque to x-rays (*angiography* or *angiocardiography*). By comparison with these studies performed on the normal, it is now possible to diagnose even small congenital abnormalities.

It is no longer necessary to guess about the exact nature of congenital disorders. Now we can demonstrate not only the sites of the disturbances, but also their severity in terms of disordered function. We have learned that murmurs alone may be misleading, and that severe stresses upon the heart may be evident only with this precise information and when enlargement of the heart is not apparent by the conventional x-ray techniques. Furthermore, the use of dyes and of safe radioactive substances injected into the circulation, with measurement of their distribution by modifications of the Geiger counter, may yield important evidence concerning congenital abnormalities.

It is possible to measure the effects of congenital disturbances, over considerable periods of time, by repeating these tests periodically. Thus a child may not be in need of operation at

the age of five, but repetition of the tests five years later may show that irreparable damage to the heart and lungs will occur if operation is further deferred.

By emphasizing the role of advances in the techniques for the clarification of congenital defects I should not wish to indicate that only special tests are useful. As with other types of heart disease, the diagnosis is a well-rounded approach, and is formulated according to the Five-Point Quiz as discussed in Chapter II. In addition, however, the possibility of surgical correction, for both rheumatic and congenital heart disease, makes it mandatory to employ all measures available for enhancing diagnostic accuracy.

Blood Vessels Outside the Heart

As pointed out in the summary of the development of operations upon the heart and blood vessels (Chapter V), two procedures have by now become commonplace. These concern those disorders of the blood vessels which do not produce cyanosis, or blueness of the skin and nail beds. Among these, two have been found to be diagnosed readily and can be corrected by operations performed within the chest but outside the heart.

The *ductus arteriosus* conducts blood from the aorta to the pulmonary artery prior to birth, acting as a by-pass around the lungs. Although this channel usually closes shortly after birth

it may remain open throughout childhood and into adult life. Characteristically its persistence as an open channel is indicated by a whirring, machinery-like murmur heard through a stethoscope, but the more precise methods described above may be required to detect its presence and to evaluate the magnitude of the disturbance caused by it. Not infrequently the persistence of the ductus may mean that fifty per cent of blood leaving the left ventricle may go back into the lungs, instead of to the head, trunk, and limbs. This involves extra work for the heart to maintain adequate blood flow, and may result in gradually increasing heart failure. Furthermore, infection may localize in the ductus and may threaten life by discharging bacteria throughout the body. Thanks to new antibiotic drugs such infections now can usually be controlled. It is evident, however, that prevention is better than cure. To avoid the dangers of heart failure or infection of the ductus, operative closure of the ductus is usually advisable during early childhood when it is evident that a sizable shunt is present. Thanks to advances in diagnosis, anesthesia, and surgery the operative correction of patent ductus can now be made with little more risk than the removal of an appendix.

A second common congenital abnormality of a blood vessel is a narrowing of the aorta in the region of the ductus. This constriction is termed *coarctation of the aorta*. It may be an almost

complete closure, with only a pinpoint channel remaining. This results in an elevation of blood pressure in the arteries of the head and arms, and a very small pressure in the arteries of the abdomen and legs below the coarctation. The chief danger to the patient is that damage to blood vessels in the head and eyes may eventually follow the increased blood pressure in the upper part of the circulation, as in hypertension from other causes.

To avert the possibility of blindness or a stroke, operations for the relief of coarctation have been devised. If the area of constriction is small a segment of the aorta can be removed and the two ends rejoined directly. To perform this operation successfully it was necessary to learn how to sew together the edges of blood vessels so that leakage would not occur when considerable pressure from within the artery was exerted on the area. This involved much animal experimentation. If the operation could not be performed in a few minutes, damage to the nerves and vital organs below the coarctation would result from interruption of their blood supply. Accordingly, for prolonged operations one of two safeguards can be used: (1) A shunt or graft can be inserted temporarily around the area of coarctation. Either human tissue (artery or vein) or a plastic tube can be utilized for this shunt. After the coarctation is corrected the shunt is removed. (2) The needs of

the body for oxygen can be reduced during the operation by lowering the body temperature (*hypothermia*) through the use of ice bags or other refrigerants. It need hardly be added that much experimentation was required before these two techniques were perfected for use in humans. With respect to material for shunts, it had to be demonstrated which materials were most acceptable to the body, could be sterilized most readily, and would have the properties of durability and elasticity most similar to the human aorta. For hypothermia to be successful it had first to be shown how various tissues react to lowering the body's temperature, what measures were most safe and practical for refrigeration, and what body temperatures served best to diminish the need for blood and oxygen during operation. It was learned, among other things, that the heartbeat is considerably slower during hypothermia, and that nerve and muscle needs for oxygen are much diminished. The techniques of shunting and hypothermia are also employed during operations within the heart, to be described shortly.

Other congenital anomalies of the blood vessels outside the heart have been demonstrated by catheterization and angiocardiography. These may be persistent prenatal vessels which are the counterpart of the arterial rings seen normally in fishes or lower animals. When they persist in the human they may interfere with swallowing or

other functions. A fairly common abnormality is a free communication between arteries and veins, or between arteries normally having a large differential in pressure, as in the case of the ductus arteriosus. This may occur within the lung. Such shunts usually place an unusual strain on the heart and deserve surgical correction to prevent or correct heart failure. Usually cyanosis is not evident in association with these communications.

A type of blood-vessel anomaly most serious to the individual is one in which blood vessels enter or leave improperly certain chambers of the heart. If, for instance, oxygenated blood from the lung (*pulmonary*) veins enters the right atrium there will be abnormal admixture of blood in a chamber properly containing only blood low in oxygen content. Perhaps the most severe abnormality of this type is seen when the aorta leaves the right ventricle and the pulmonary artery leads from the left ventricle (*transposition*). In this situation there is marked cyanosis, since blood low in oxygen content (*unsaturated*) is pumped directly to the head, limbs and trunk. Oxygenated blood from the lungs is circulated back to the lungs. Unless shunts coexist inside or outside the heart, as is usually the case, babies with transposition do not long survive. Because of advances in diagnosis and surgical technique even the severe handicap imposed by transposition can be alleviated.

Anomalies Within the Heart

Congenital malformations within the heart are basically few in type: (1) Chambers may be too large or too small to function properly. (2) One or more of the valves of the heart may be too tightly closed (*stenotic*), or too widely open (*regurgitant*). (3) There may be an abnormal communication between the left and the right auricle (*interatrial septal defect*), or between the left and the right ventricle (*interventricular septal defect*). These possibilities allow one to visualize the more common abnormalities. As its name suggests, the *tetralogy of Fallot* has *four* parts: (1) Stenosis of the pulmonary artery, (2) an interventricular septal defect, (3) thickening of the walls of the enlarged right ventricle, and (4) overriding of the aorta so that it receives blood simultaneously from both the left and the right ventricles. The tetralogy is one of the commonest types of congenital anomalies producing *cyanotic* or blue babies.

What Is Cyanosis?

Cyanosis, or blueness of the skin and nail beds, is evidence that insufficient oxygen has been taken up by arterial blood. In congenital heart disease this can occur if there is admixture of blood between both sides of the heart (*interatrial or interventricular septal defects*), or if there is inadequate flow of blood through the lungs due

to stenosis of the pulmonic valve or to a very small pulmonary artery. It is evident that both conditions exist when the tetralogy of Fallot is present.

Cyanosis indicates serious interference with the nutrition of the tissues of the body. The growth and development of such children is often impaired. In the case of Jimmy, Chapter I, the diagnosis was tetralogy. His response to operation was dramatic. Yet cyanosis alone is not a reason for operation in that some persons with cyanotic congenital heart disease have been known to live well beyond middle age. The overall picture, including periodic physical examinations and the application of precise tests, serves to indicate whether or not operation should be attempted, and when.

What Types of Operation Can Be Done?

We have already touched upon some of the surgical procedures which can be performed to correct various types of heart disease, such as the use of synthetic grafts for blood vessels. We have also considered how advances in the use of anesthesia and of lowering the body's temperature (*hypothermia*) have permitted surgeons to work on and within the heart. One of the practical difficulties to be overcome has been the need for the surgeon to see structures within the heart, without their being obscured by blood. To over-

come this difficulty it was first necessary to devise machines to take over the functions of the heart and lungs. This has been done, following years of meticulous research. The second problem was to stop the heart from beating, temporarily, with the assurance that it could be started again after the surgical repair was completed. Through the use of the drugs acetylcholine or potassium citrate this has been accomplished. Now the surgeon can place his stitches (*sutures*) accurately while the inside of the heart lies exposed, free from blood, and quiet. When his work is completed the heart beat can again be resumed. This is indeed a modern miracle.

The actual types of repair possible can be visualized in relation to the nature of various congenital anomalies. If a valve orifice is too small (*stenotic*), its opening can be enlarged by delicate cutting operations. If the valve opening is, contrarily, too large (*regurgitant*), it can be made smaller by encircling the base of the valve with a constricting band, or by other corrective techniques. These measures have proved effective, whether the valvular disease be of congenital or rheumatic origin.

Where one or more of the four chambers of the heart is too large or too small, improvement can be expected only when causative valvular or blood vessel abnormalities can be corrected. For instance, if the left atrium is markedly dilated due to mitral stenosis, the operation enlarg-

ment of the mitral valvular orifice may result in proper emptying of the atrium, which will then tend to assume a more normal size. Not infrequently, in this situation the surgeon may remove a portion of the dilated atrium at the time he enlarges the mitral valvular orifice.

With the advent of "open-heart" surgery, the heart being at a standstill, defects between the atria and the ventricles can now be closed, using patches of plastic or of human tissues. Even prior to the development of open-heart techniques interatrial septal defects were closed successfully, with or without opening the thin walls of the atria. As regards the latter, it was discovered that the walls of the atria could be cleverly tucked in to close the defect without opening the chambers. Because the interventricular septum is situated deeply between thick-walled cavities, corrective operations can be performed properly at present only under direct visualization.

The Artificial Heart-Lung Machine or Pump-Oxygenator

The way in which a heart-lung machine is attached to the human circulation is shown in Diagram 3. Four types of pump-oxygenators are currently in use in the United States. The *screen oxygenator* devised by Dr. Gibbon of Philadelphia was the first to be used successfully in operations upon patients. A thin film of blood is allowed to trickle down a wire screen in an en-

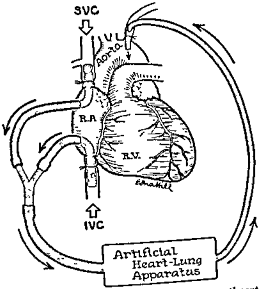


Diagram 3. How the pump-oxygenator (*heart-lung machine, cardio-pulmonary by-pass, extra-corporeal circulation*) is adapted to take over the functions of the heart and lungs during open-heart operations. S. V. C.—superior vena cava. I. V. C.—inferior vena cava R. A.—right atrium. R. V.—right ventricle. Blood is collected from the great veins (venae cavae) entering the right atrium by inserting tubes into the superior and interior venae cavae. After being oxygenated artificially, it is pumped mechanically into the aorta.

closed case containing ninety-seven per cent oxygen and three per cent carbon dioxide, just as rainwater forms a film on a window screen during a rainstorm. Although highly effective as an artificial lung, and as a heart also when a suitable pump is included, this apparatus is expensive and difficult to clean and to sterilize.

A *bubble oxygenator* has been used successfully in many patients, serving also as the lung part of a heart-lung machine. Here gaseous oxygen is bubbled through a column of venous blood.

A *rotating disc oxygenator* was first developed in Sweden. It involved the use of 100 small vertical discs on a horizontal axle. As these discs dipped into a pool of venous blood a thin film of blood formed on the surfaces of each disc and was exposed to a high concentration of oxygen. With this machine four pints of venous blood could be oxygenated per minute.

A fourth type of oxygenator was developed in Cleveland. This *membrane-type apparatus* consists of a number of sheets of synthetic Teflon over which blood flows as in the case of the screen oxygenator, exposing a thin film of blood to the surrounding oxygen.

In designing pumps to reproduce the heart's action consideration must be given to simplicity of construction, reliability, ease of cleaning, and a sufficiently gentle action to avoid unnecessary injury to the red blood cells. A successful roller-type pump was devised in the laboratories of

Dr. Michael E. De Bakey in Houston, Texas. Blood flow is gently accelerated through a plastic tube by the external pressure exerted by a series of rollers.

Another practical problem, which relatives of a patient for whom the use of the pump-oxygenator is planned must share with the doctors, is the necessity to have sufficient blood on hand to fill the pump oxygenator. This need can be met, despite the inconvenience and expense involved. A number of blood donors will be called upon.

CAUTION! OPERATION IN PROGRESS

To witness an operation upon the heart, with the use of the pump oxygenator, is to see the fruits of many years of experimentation applied toward the improvement and safety of a single patient. Some of the personnel and equipment can here be described.

There will be a chief surgeon, with at least two assistants working closely with him. At least four skilled nurses will be a part of the team. One or more highly trained physician anesthetists will be in attendance. Other physicians, to a total of twelve or thirteen, may be required to operate special apparatus. All personnel will wear sterile caps, gowns, gloves and masks. All instruments, drapes, packs and bandages will have been sterilized and will be handled with sterile precautions.

Only blood freshly drawn from donors will be

used to load the pump oxygenator. It will be rendered free-flowing and deprived of its usual tendency to clot through the use of an agent long since discovered by physiologists and known as *heparin.*

As the surgeon prepares the shunts required for the heart-lung machine (Diagram 3) one will be impressed by a variety of mechanical and electrical equipment reminiscent of the cockpit of a modern high-speed airplane. The pump-oxygenator is a complicated unit by itself, as is the machine for deliverying various anesthetic agents. An impressive instrument panel will allow the surgeon, anesthetist, and the physicians in charge of the pump-oxygenator to read at a glance such important information about the patient as (1) pressures in his arteries and veins; (2) the pattern of the electrocardiogram, and the brain waves (*electroencephalogram*); (3) body temperature; and (4) flow of blood through the pump-oxygenator. Gauze packs and sponges will be weighed in the operating room suite to allow an estimate of blood loss.

In an atmosphere of quiet orderliness each member of the team will pursue his duties. The preparation for the use of the pump-oxygenator is meticulous and time-consuming. When it is functioning properly and the patient's condition is satisfactory the heart's action is arrested, the heart is opened, and the operation inside the heart is performed expeditiously. After the heart

is closed the oxygenator is not disconnected until it is clear that the heart has recovered sufficiently to carry on without assistance. Then the shunts are removed, the blood vessels closed, and small bleeding points are secured finally before the chest wound is closed in layers.

Throughout the days and nights to follow, the same care is bestowed upon the patient by a team of skilled doctors and nurses. Having utilized the benefits of many years of experience and experimentation in preparing for and pursuing the operation, his doctors take no risk that any complication, improperly handled, might allow all this work to become nullified, to the detriment of the patient.

This view of the operating room will, I hope, persuade you of our concern for our patients and their loved ones. Open-heart surgery is in its infancy, with many problems yet to be solved. For their solution, doctors are working with other highly trained scientists including physicists, chemists, and engineers. Apparatus devised to promote the safety and success of such operations is under close scrutiny in order that it can be improved and simplified when possible. We anticipate that the future will see continued substantial advances in this field.

Chapter VII

WHAT IS THE WORTH OF A HUMAN LIFE?

The Doctor's Decision

After a man dies, there are those charged wit evaluating the monetary worth of his estate in order to apportion it according to legal requirements. Few would disagree that the sum involved bears little relationship to the deceased's actual worth to his family and his community. An attempt to measure the true worth, comparatively, of different individuals likewise leads to unanswerable questions. Is the life of a policeman who saves a talented pianist, threatened by thugs, of more or of less value than that of the pianist? To repeat: it is easy to measure the monetary worth of an individual, or to measure the contributions he has made to society. His net value to his friends and family can at best, however, be estimated only roughly. How does a doctor estimate the worth of his individual patients?

Our forefathers, and many since, have dedicated their lives to the proposition that we are all created free and equal. The doctor's creed goes two steps further: 1. A human life is of

infinite value, and is the most precious thing on earth. 2. All human lives are equally valuable and are to be conserved and defended to the end. It follows that if several courses of action are available, the doctor must decide which one is most likely to preserve life and to improve health.

Through being untroubled by the comparative worth of various individuals, doctors have found a place in society which has evoked the tribute of Robert Louis Stevenson.

> There are men and classes of men that stand above the common herd: . . . the physician almost as a rule. He is the flower (such as it is) of our civilization. Generosity he has, such as is possible to those who practice an art, never to those who drive a trade; discretion, tested by a hundred secrets; tact, tried in a thousand embarrassments; and what is more important, Heraclean cheerfulness and courage.
>
> From the dedication to *Underwoods* (1887)

By thus expressing his gratitude to physicians Stevenson has also reminded them of their obligation to adhere to their principles of ethics.

Second only to his devotion to his patients, and coupled with a desire to give of his best to improve their health and happiness, is the physician's dedication to the principles that: (1) He should aid in the professional advancement of his colleagues in all possible ways, and (2) he is obligated to increase the sum total of medical knowledge. In the foregoing chapters we have

frequently discussed the benefits from *research* which have accrued to patients with heart disease. Since the teaching of medical students and doctors would be static without research, it is appropriate to pause here to answer some of your questions concerning this important part of the growth of medical knowledge. To do so will, I believe, give you a better understanding of how discoveries are made, such as those allowing operations to be performed on the heart, and many other improvements in the treatment of patients. Indeed, it will explain the basic techniques through which all important advances in medicine are achieved.

Who Engage in Medical Research?

Perhaps you have found yourself curious as to the way in which medical research is performed. Most of you have had blood and urine samples taken in a hospital or in a doctor's office. Is this a type of medical research? It is, to the extent that all physicians feel it an obligation to share important discoveries with their colleagues. Even simple tests of this nature may lead to new knowledge about disease. This knowledge may be shared in informal discussion, presented as scientific papers at medical meetings, or may lead to formal reports published in scientific journals or textbooks. To this extent all doctors share in medical research.

As one who has been connected with a medi-

cal school throughout his professional life, the author has seen the picture in sharp focus: Doctors who are most productive in research have a strong and sustained desire to contribute to advances in medicine, based on a long background of scientific training. Often an interest in science or natural history has been theirs since childhood. Stimulated by parents and teachers they have prepared themselves by attending college, taking the prescribed medical course of four years, serving an internship for a year or two, and have then done intensive work in a special field for another *two* to *ten years.* Some decide to make research their full-time occupation. Others may continue to pursue research part-time in connection with practice and teaching.

Throughout their professional lives they receive advice and help from their associates. They have access to libraries from which they can learn of medical activities throughout the world.* Now you are beginning to see the picture as a teacher in a medical school has repeatedly observed it: Give the well-prepared young doctor an opportunity to work in a field which is exciting to him,

* The author can recall drawing up a list of 100 references to be reviewed in preparation for a report concerning a disease rare in this country but common in the tropics. Many foreign journals were involved. Upon presenting this list of 100 medical journals to the Library of the College of Physicians in Philadelphia, he was able to find ninety-eight of them in this library. The remaining two were easily obtained on loan from medical libraries elsewhere.

and he will invariably produce something worth while for the advance of medical knowledge. He may not expect his financial return to equal that of his classmates in college who entered the business world while he was still in medical school. His sponsors, however, must pay him a living wage if he is on a full-time research program, or allow him to supplement his income through practice or other means. He will rightfully expect professional recognition in keeping with the character of his work.

The Training of an Investigator: A Case Report

To illustrate the background of a young medical scientist currently performing research in heart disease the following *curriculum vitae* is reproduced. His name is fictitious and the names of institutions concerned have generally been deleted, but dates are preserved.

Name: John A. Doe, M.D.
Born: May 9, 1926.
Married: March 26, 1955.

Education:

High School, graduated 1943.
Attended —— University, June-July, 1943 (freshman in engineering).
Attended State College of Agriculture and Engineering, July, 1943-July, 1944 (mechanical engineering).

State College, December, 1944-June, 1945 (ASTP mechanical enginering).

Attended —— College, December, 1946-September 1947 and summers of 1948 and 1949 (liberal arts).

Attended State College, September, 1947-February, 1949, receiving B.S. in Physics.

Attended University of ——, March-May, 1949 (pre-med. courses).

Attended —— School of Medicine, 1949-1953, M.D., June, 1953.

Military Service:

Enlisted Reserve Corps, July, 1943-July, 1944, in college.

Active Duty: Infantry, August, 1944-December, 1944, Camp ——.

ASTP, December, 1944-June, 1945.

Manhattan District, Corps of Engineers. Detached to work for —— Corp., Oak Ridge, Tenn., July, 1945-March, 1946. Bikini Bomb Tests, March, 1946-August, 1946.

Discharged August, 1946, Rank T-3, M.O.S. mechanical engineer.

Employment and Miscellaneous.

Licensed amateur radio-operator, Station ——, 1941.

Instrument Engineer for —— Corp. in the Gaseous Diffusion Plant, Oak Ridge, Tenn., July, 1945-March, 1946.

Member Radiological Safety Section, Crossroads Operation, March, 1946-August, 1946 (Oakland, Berkeley, and San Diego, California, and Bikini).

Field Engineer for —— Instrument Company,

August, 1946-December, 1946 (Cleveland, Kwajalein).

Electronics Technician, then Junior Engineer, ——College, part time while in school 1948, full time to March in 1949.

Worked in English boys camp and in the London Hospital, summer of 1950.

Research with Dr. ——, University of ——, Physiology Department, summers and school years 1951-53; July, 1954-June, 1955.

Intern, Hospital of ——, July, 1953-June, 1954.

Assistant Resident in Surgery, University of ——, July, 1953-June, 1954.

Instructor in Physiology, University of ——, School of Medicine, July, 1954-June, 1955.

Will complete formal surgical training July, 1960.

Societies:

American Physical Society.
Sigma Pi Sigma (physics).
Pi Mu Epsilon (mathematics).
—— Physiological Society.
Candidate group, American College of Surgeons

Publications:

Co-author of seven scientific papers 1952-1959.

With this background we feel this young man is capable of making scientific observations of real value, and of performing well as a surgeon. He envisions a career in the department of surgery of a medical school. Modern schools of medicine encourage applications for admission from such young men with varied backgrounds.

Who Pays the Bills?

Who pays for the laboratory equipment and instruments used by the investigator in his research? In the last analysis, you and I do. We benefit from his discoveries, and should contribute to the cost of them. Each year millions of dollars in research grants come from various sources. Individuals contribute large or small amounts directly. Various agencies of our government sponsor research, to be performed in hospitals or medical schools throughout the country, or in their own hospitals or laboratories. These include the Department of Health, Education, and Welfare, and various departments of the Army, Navy, and Air Force. Large philanthropic funds and foundations have supported many important advances. Among these are the Life Insurance Research Fund, the Commonwealth Fund, the Josiah Macy Foundation, and others too numerous to mention. Manufacturers of drugs have supported medical research generously. According to a statement issued by the Pharmaceutical Manufacturers' Association, dated January 30, 1959, the federal expenditure for medical research was $227 million in 1958. During the same year it is estimated that the pharmaceutical industries invested in excess of $170 million in medical research.

Because of the need to provide funds adequate for continued programs of research, organizations

such as the American Heart Association have maintained an aggressive and fruitful annual series of fund-raising campaigns. From funds so obtained a large variety of research projects are being supported. These range from projects for high school students in science, to a guarantee of *lifetime support for the individual and his laboratory costs* in the case of a few "career" investigators.

In a sense this little book is an off-the-record progress report from your medical profession, with respect to research in the field of heart disease. You have helped us to do the job. We hope that you are pleased with our achievements. The costs have not been high, when viewed either in terms of lives saved, or when compared with other of our expenditures. We must continue to apply the instruments of modern science to the saving of lives. No parent in America today has sufficient wealth to write a check for the total cost of developing, over the years, the techniques required to save the life of his child who may require intensive medical and surgical care for the correction of his heart disease. We must each share the responsibility for continuing research in heart disease, since each of us will enjoy its benefits, directly or indirectly.

INDEX

R

S

T

U